THE MATURATION OF CORTICAL AUDITORY EVOKED POTENTIALS IN CHILDREN WITH NORMAL HEARING AND HEARING IMPAIRMENT

A thesis submitted in partial fulfilment of the requirements for

the Degree of

Master of Audiology

in the Department of Communication Disorders

University of Canterbury

2008

By Jennifer Walker

Table of Contents

List of Figures

List of Tables

Acknowledgements

I wish to express sincere appreciation to my supervisors, Greg O'Beirne and Valerie Looi. Your constant patience and support throughout the entire process reminded me that this was achievable. I especially appreciate you answering my questions over and over again. I especially appreciate you answering my questions over and over again. I especially appreciate ...

Thank you to Neil Heslop, Shirley Wintersgill, and the entire team at the Southern Cochlear Implant Program. Your invaluable input and support ensured this thesis got underway.

Thanks to Michael Weavers, from the Department of Civil and Natural Resources Engineering, for his technical assistance in helping to adapt our IHS system to deliver free-field stimuli.

Importantly, I would like to thank all the children and their families who took the time out to come and watch a movie with me. Just remember: "In the land of Joke-a-lot, everybody laughs a lot, don't have to work all day, just have to joke and play."

Finally, heartfelt thanks to all my family for believing in me and encouraging me along. But above all to the two men in my life: Matt, my husband and rock, and David, my son and constant excuse for a study break.

Abstract

Cortical auditory evoked potentials (CAEPs) are a non-invasive tool that can provide objective information on the functioning of the auditory pathways. As a result, there is increasing use of these measurements to support the early implantation of cochlear implants in children. However, the maturation of CAEPs in children who have received cochlear implants is still not well understood. This study investigated five children who received cochlear implants prior to 3.5 years of age and compared their CAEP waveforms to five normal-hearing children who were matched for age and five normal-hearing children who were matched for time-in-sound. In addition, a case study was completed, comparing an age-matched child who received hearing aids prior to 3.5 years of age to one of the children with cochlear implants. The latencies and amplitudes of the positive and negative peaks (P1, N1, P2, and N2) of the CAEP waveforms were measured and analysed using statistical techniques, including one-way repeated measures ANOVA. Our hypothesis was that children who received cochlear implants prior to 3.5 years of age would have CAEPs similar to their age-matched peers with normal hearing. Additionally, it was expected that the two matched children with hearing instruments would have CAEP waveforms that are similar. The measurements recorded demonstrated no conclusive results between the children with cochlear implants and either of the normal hearing groups. In the case study, the latencies and amplitudes of the child with a cochlear implant were similar to those of the child with hearing aids, supporting the second hypothesis. This study highlights the need for further research on a larger scale in order to more clearly determine the manner in which CAEP waveforms mature in children with cochlear implants.

Abbreviations

µV:	microvolts
AEP:	auditory evoked potentials
ALR:	auditory late responses
ANOVA:	analysis of variance
BAEP:	brainstem auditory evoked potentials
CAEP:	cortical auditory evoked potentials
CI:	cochlear implant
dB:	decibel
dB HL:	decibel hearing level
dB SPL:	decibel sound pressure level
HA:	hearing aids
Hz:	Hertz
IHS:	Intelligent Hearing Systems
ISI:	interstimulus interval
LLAEP:	long latency auditory evoked potentials
m:	months
MLAEP:	middle latency auditory evoked potentials
MMN:	mismatch negativity
ms:	milliseconds
NH:	normal hearing
TiS:	time-in-sound
TV:	television
y:	years

Chapter 1

Introduction

1.1 Introduction

Cortical auditory evoked potentials (CAEPs) are a non-invasive, objective measure that can provide detailed information on the central auditory nervous system. The ascending pathway of the auditory nervous system starts at the cochlea in the inner ear, travels along the auditory nerve and passes through a number of nuclei before reaching the auditory cortex in the cerebrum, as shown in Figure 1 (Calhoun, 2008; Musiek & Oxholm, 2000; Rappaport & Provençal, 2002).

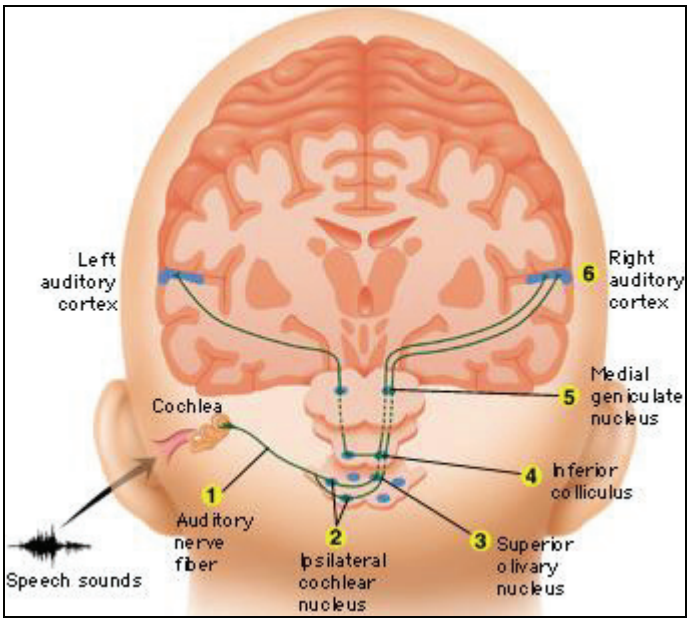


Figure 1: A simplified representation of the auditory pathway, when excited unilaterally via the left cochlea neural responses, pass along the ipsilateral auditory nerve (1), and then through the ipsilateral cochlear nucleus (2) and the contralateral superior olivary nucleus (3), before being passed through the lateral lemniscus to both the ipsilateral and contralateral inferior colliculi (4) and medial geniculate nuclei (5), to terminate in the left and right auditory cortices (figure from Calhoun, 2008).

The first part of the central auditory nervous systems is the cochlear nucleus complex which consists of the anteroventral cochlear nucleus, posteroventral cochlear nucleus, and dorsal cochlear nucleus. These three nuclei appear to work independently and process neural information on timing, onset, duration, and periodicity (Rappaport & Provençal, 2002). The anteroventral cochlear nucleus and posteroventral cochlear nucleus both project to the superior olivary complex which is comprised of the lateral superior olive and the medial superior olive as well as the medial nucleus of the trapezoid body. The superior olivary complex amalgamates incoming information from both the ipsilateral and bilateral cochlear nucleus complex, indicating that it has a role in sound localisation (Rappaport & Provençal, 2002). The fibres then pass along the lateral lemniscus to the inferior colliculus. All ascending fibres pass through the inferior colliculus which appears to integrate and organise all the auditory information (Rappaport & Provençal, 2002). The medial geniculate body located in the thalamus is the final nucleus that the auditory pathway passes through before reaching the auditory cortex. The auditory cortex appears to process complex combinations of information. These may include the analysis of complex sounds, localisation, perception of temporal patterns, and identification of auditory stimuli rather than detection (Rappaport & Provençal, 2002).

An auditory evoked potential is an electrical response recorded in the auditory nervous system in response to an auditory stimulus. A variety of stimuli, including clicks, pure-tones, and speech sounds, can be used to elicit auditory

evoked potentials. The electrical activity that occurs as a result is recorded via electrodes placed on the scalp and/or ears. Due to the small size of the evoked potential relative to the background noise, multiple waves must be recorded and averaged in order to improve the signal-to-noise ratio and allow a clearly defined waveform to appear. Furthermore, the waveforms, and in particular, the peaks must be replicable to ensure the reliability of the recordings (Hall, 1991).

Auditory evoked potentials can be recorded at various stages along the auditory pathway, and have therefore been subsequently divided into short, middle, and long latency evoked potentials depending upon the delay between the presentation of the stimulus and the resultant electrical signal (Burkard & Secor, 2002; McPherson & Ballachanda, 2000). CAEPs occur at least 50 ms following acoustic stimuli presentation, occurring later than both the auditory brainstem response (<10 ms) and the middle latency responses (10 – 50 ms) as shown in Figure 2. They are, therefore, referred to as long latency AEPs (LLAEPs) or auditory late responses (ALRs) (Burkard & Secor, 2002; McPherson & Ballachanda, 2000).

The LLAEPs occur between 50 and 300 milliseconds (ms) after stimulus onset (Wunderlich & Cone-Wesson, 2006). In adults, the waveform morphology is dominated by a well defined negative peak, N1, which typically has a latency between 90 and 150 ms post-stimulus onset. This is preceded by a smaller positive peak, P1, which occurs at approximately 50 ms post-stimulus onset. N1 is

also normally followed by P2, a broader positive peak, at about 175-200 ms post-stimulus onset (Purdy et al., 2001; Wunderlich & Cone-Wesson, 2006).

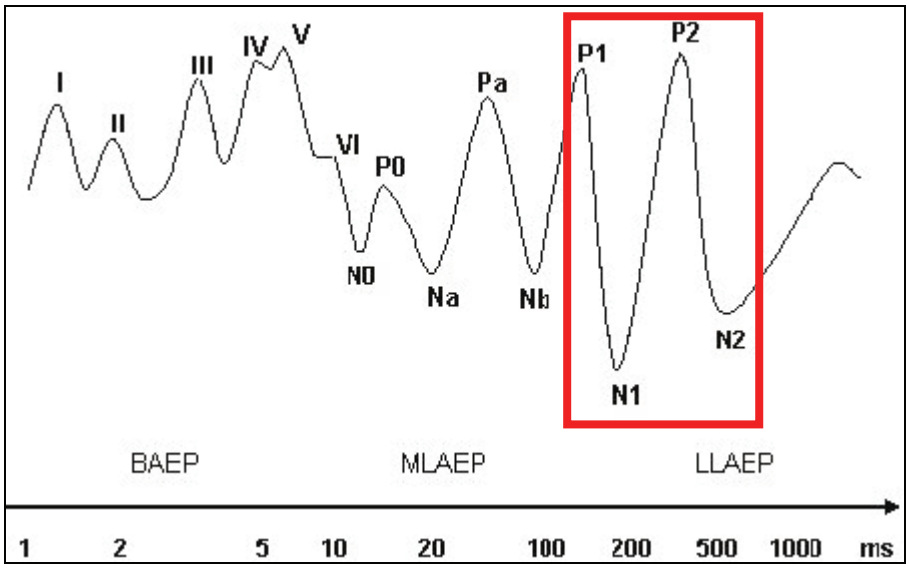


Figure 2: Auditory evoked potentials are divided into early or brainstem auditory evoked potentials (BAEP), middle latency auditory evoked potentials (MLAEP), and long latency auditory evoked potentials (LLAEP). The marked area highlights the four main wave components of the long latency or cortical auditory evoked potentials (figure adapted from Danmeter, 2006).

It has been known that sensory inputs, such as acoustic stimuli, can modify cortical electrical potentials since Caton's experiments on rabbits in the late 19th century (Caton, 1875). It was first noted in 1939 that electroencephalography recordings contained a component dependent upon acoustic stimuli (Davis, 1939). As maximum amplitude of the potential is recorded when the electrodes are placed on the vertex, it was originally believed that the waveforms represented a

nonspecific cerebral process. However, further research and developments in technology have enabled more precise recordings, confirming the presence and increasing the clinical applications of LLAEPs (Stapells, 2002).

These LLAEPs are believed to reflect activity of excitatory post-synaptic potentials at the level of the thalamus and higher auditory cortex (Ponton & Don, 2003; Purdy, Kelly, & Thorne, 2001; Sharma & Dorman, 2006; Wunderlich & Cone-Wesson, 2006). Each peak of the CAEP waveform appears to originate from multiple neural generators (Ponton & Don, 2003; Sharma & Dorman, 2006). The main generators appear to originate from within the auditory cortex bilaterally. However, there appears to be some contribution from the modality nonspecific areas (Stapells, 2002). As the CAEP waveform is generated from within the auditory cortex it is believed that a recording reflects the fact that the acoustic stimulus has been perceived by the listener. The sound level at which a CAEP is first measurable correlates closely with a subject's psychophysical threshold (Stapells, 2002). This means the acoustic signal must have traversed the entire acoustic pathway in order to be recorded. Therefore, the latencies of the peaks reveal the entire synaptic transmission time, including any delays in the transmission time along the auditory pathway or cortex (Ponton & Don, 2003; Sharma & Dorman, 2006).

1.2 Categorisation of CAEPs

CAEPs can be classified as either discriminative or obligatory (Purdy et al., 2001; Wunderlich & Cone-Wesson, 2006). Discriminative, or endogenous, CAEPs, such as the mismatch negativity (MMN) and P300, are elicited using an "odd-ball" paradigm, requiring the listener to have the ability to discriminate between changes in the stimulus (Purdy et al., 2001). In contrast, the elicitation of obligatory, or exogenous, CAEPs is solely dependent on the external stimulus and the integrity of the central auditory nervous system (Wunderlich & Cone-Wesson, 2006). As a result, obligatory CAEPs are less affected by non-auditory factors, such as attention and memory, and can therefore be recorded reliably in all population groups, including infants and young children (Wunderlich & Cone-Wesson, 2006).

Obligatory CAEPs of adults contain two main vertex positive peaks and one main vertex negative peak, which are commonly called P1, P2, and N1 respectively (Purdy et al., 2001). One other negative peak, N2, frequently follows P2, as shown in Figure 3 (Wunderlich & Cone-Wesson, 2006). The response represents summed activity from a number of generators in the auditory nervous system. In particular, N1 is believed to reflect activity of at least four neural generators, while P2 is thought to primarily reflect activity in the primary auditory cortex. As these areas of activation occur at the end of the auditory nervous system, a stimulus of longer duration generates a more robust response (Purdy et al., 2001). These peaks can be elicited by a variety of acoustic stimuli, including tone-bursts,

clicks, or speech phonemes (Purdy et al., 2001; Wunderlich & Cone-Wesson, 2006).

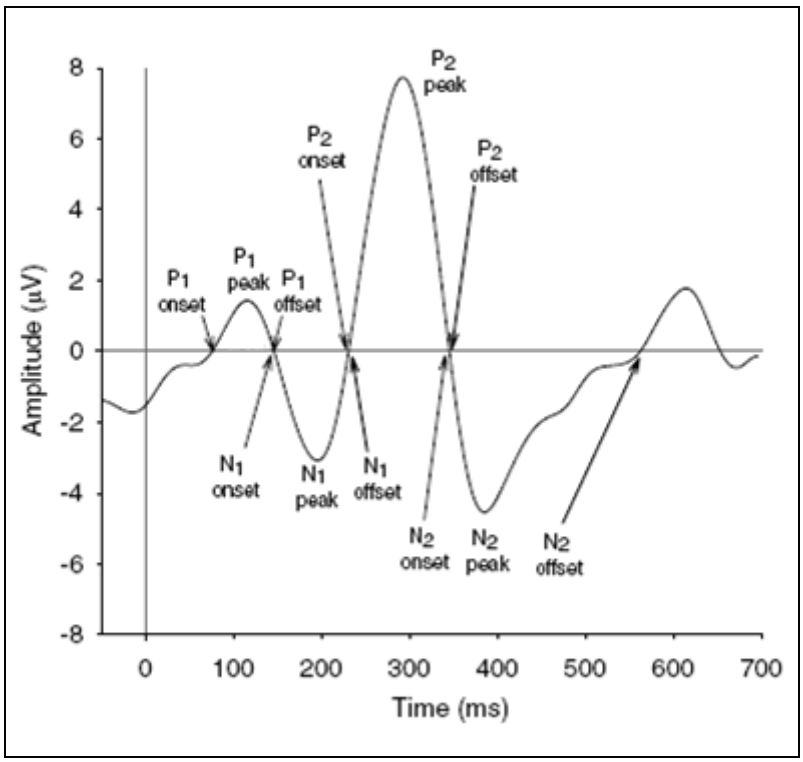


Figure 3: Cortical auditory evoked potential (CAEP) of an infant (2 years, 11 months) in response to the word "bad". Marked on the figure is the onset, peak, and offset for P1, N1, P2, and N2. Figure adapted from Wunderlich, Cone-Wesson, & Shepherd (2006).

1.3 Maturation of CAEP Waveforms

In contrast to the adult CAEP waveforms, which are dominated by N1, children's waveforms are typically dominated by P1 (Sharma & Dorman, 2006). This positive peak typically occurs at a longer latency of 100-300 ms post-stimulus onset (Sharma & Dorman, 2006).

Wunderlich and colleagues (2006) investigated the maturation of CAEPs in 49 normal hearing newborns (less than 7 days old), toddlers (13-41 months old), and young children (4-6 years old), in comparison to nine adults' evoked potential responses. They found the peak latencies of all the CAEP components were significantly shorter in adults than in children, as depicted in Figure 4. This is typical of most auditory-evoked potentials, which decrease in latency with maturational changes in myelination and synaptic efficiency. Furthermore, in general, they found that the peak amplitudes of P1 and N2 decreased with age, while the amplitudes of N1 and P2 increased (Wunderlich, Cone-Wesson, & Shepherd, 2006). These findings are in agreement with the dominant P1 present in children, and dominant N1 present in adults reported by Sharma and Dorman (2006). However, the newborn CAEP responses did not always follow the general trends. Peaks P1 and N1 were frequently absent, especially when CAEPs were evoked using pure-tones rather than speech stimulus. Peaks P2 and N2 were elicited despite the type of stimulus used, although larger peaks were recorded in response to the speech stimulus (Wunderlich et al., 2006). This may be because their CAEPs were recorded while the newborn children were sleeping, whereas all other responses were recorded from alert children and adults. It may also reflect the innate predisposition of newborns to attend to speech over other auditory stimuli (Wunderlich et al., 2006).

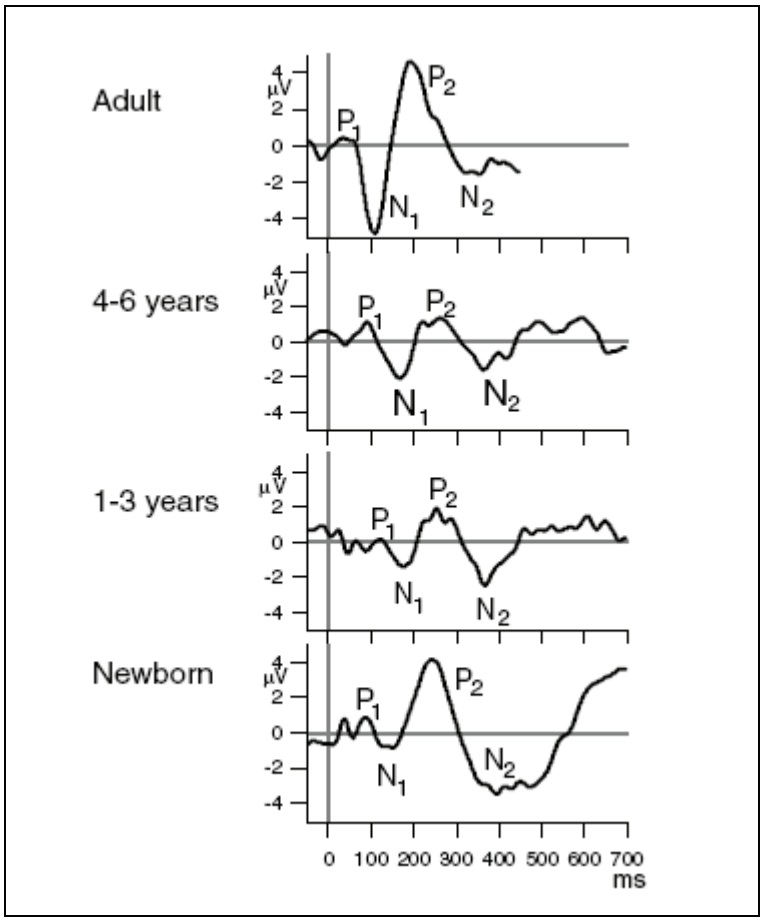


Figure 4: Maturation of cortical auditory evoked potentials (CAEP) from newborn through to infant, child, and adult waveforms. As can be seen, latency decreases with maturity, as does P1 amplitude, while N1 and P2 amplitudes increase. Figure from Wunderlich & Cone-Wesson, 2006.

The maturation of CAEPs in 86 normal hearing children from six to 15 years of age (compared to ten normal hearing adults) has also been investigated (Sharma, Kraus, McGee, & Nicol, 1997). As with the Wunderlich et al. (2006) study, the Sharma et al. (1997) study identified clear age-related changes in the P1 and N1 components of CAEPs.

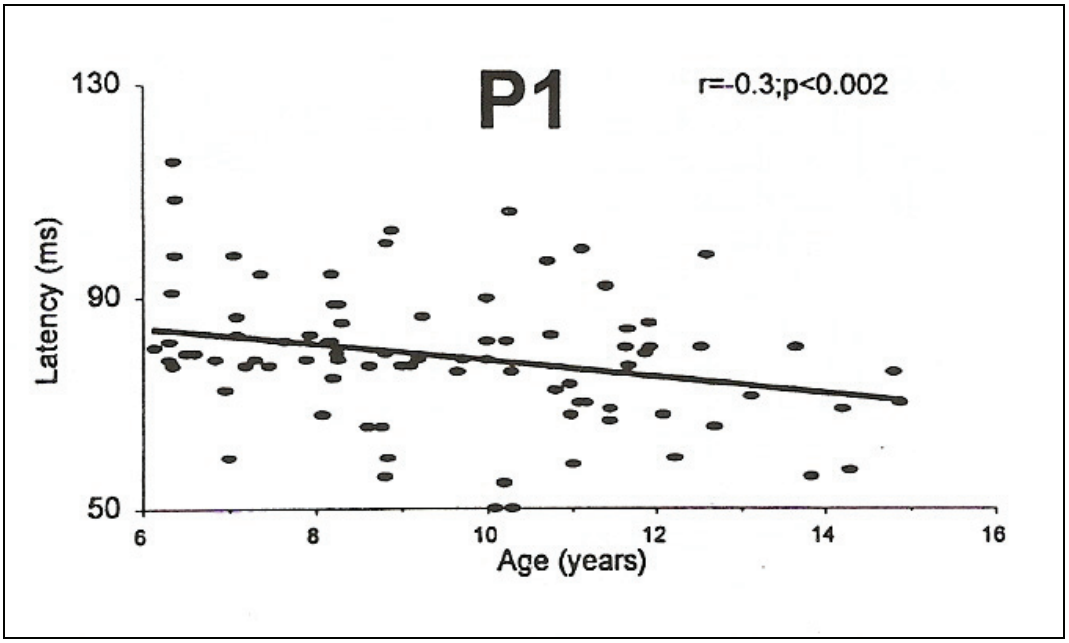


Figure 5: Decreases in latency values for P1 as a function of age for children aged from six to 15 years old. Figure from Sharma, et al. (1997).

These maturational changes included a decrease in the latencies of both P1 (as shown in Figure 5) and N1 and a decrease in the amplitude of P1 with increasing age, consistent with those findings reported by Wunderlich and colleagues (2006). In contrast, Sharma and colleagues (1997) found no age-related changes in the amplitude of N1, although they did identify a correlation between increasing age and an increase in reliability in N1 elicitation.

Sharma and colleagues (2002) further investigated the maturational changes in the latency of P1 using a broader age range (from 0.1 to 20 years of age) of 136 children with normal hearing. However, the data for over half the children

included in this study was re-used from the aforementioned Sharma study in 1997. In the latter study, they also found the latency of P1 decreases with increasing age, and that this continues until approximately 20 years of age, as shown in Figure 6 (Sharma, Dorman, & Spahr, 2002b). The changes in P1 latency occur at a more gradual rate in the second decade of life than the rapid decrease seen earlier in life (Sharma, Dorman, & Spahr, 2002b).

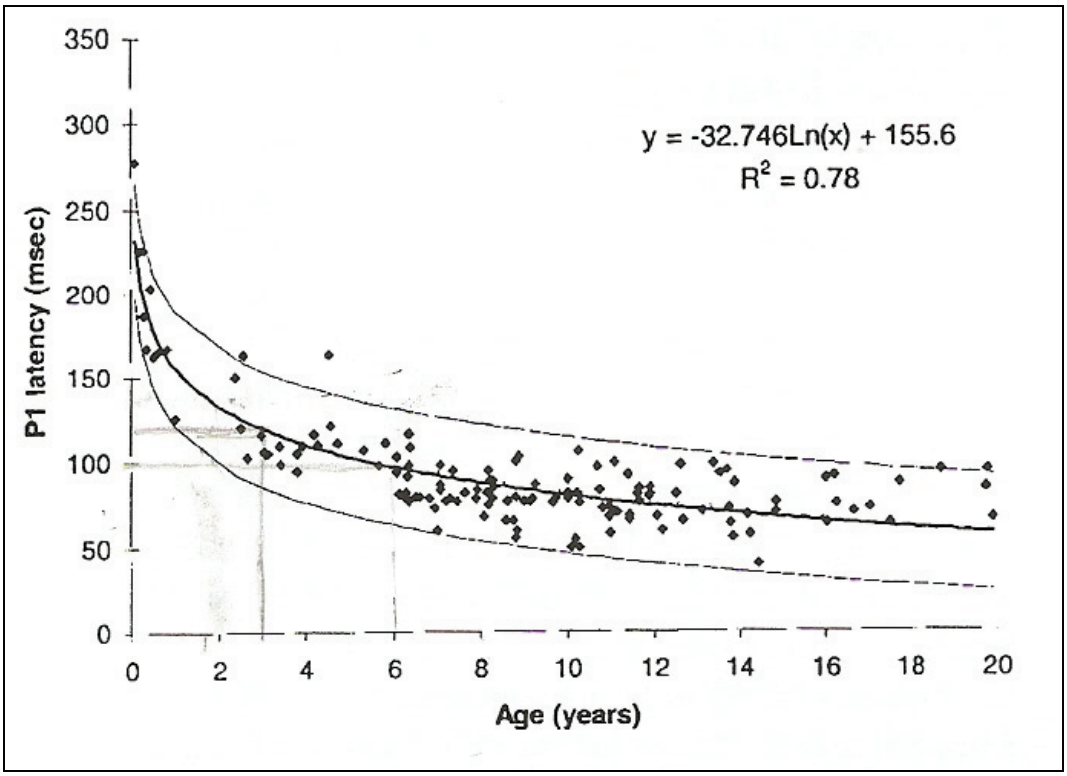


Figure 6: P1 latencies versus age function for normal hearing children. Superimposed on the raw data is the line showing the best-fit as well as the 95% confidence interval. Figure from Sharma, Dorman, & Sphar, 2002b.

The maturational changes reported in the components of CAEP waveforms appear to be consistent with the general development of the central nervous system. In

particular, the decrease seen in the latencies of all CAEP components appears to coincide with positive changes in myelination and synaptogenesis, which continue to occur through the first twenty years of life (Sharma et al., 1997). Furthermore, the different changes reported in the amplitudes and dominancy of P1 and N1 support the notion that these two peaks reflect different neural generators. N1 is believed to reflect activity in the primary auditory cortex, whereas P1 appears to reflect activity from thalamocortical sites (Sharma et al., 1997; Wunderlich et al., 2006). Therefore, recorded CAEPs indicate the stimulus has successfully ascended the central auditory pathway and resulted in activity in the auditory cortex, and for that reason, the sound was presumably heard. Evoked responses used in this manner would, therefore, provide additional, objective information on the status of the auditory system in children with a hearing impairment, as well as whether appropriate amplification is being provided (Sharma, Dorman et al., 2002b; Sharma et al., 1997).

1.4 CAEPs and Cochlear Implants

When sound is perceived by a person with normal hearing, it passes through the peripheral auditory system, including the outer, middle, and inner ears (as shown in Figure 7a), before stimulating the auditory nerve, resulting in the signal travelling to the auditory cortex via the central auditory pathway. This process is maintained even when hearing aids are used to amplify the sound, as these hearing instruments only alter the waveforms as they enter the outer ear. However, a cochlear implant alters the manner in which the auditory nerve is

stimulated. The external components of the cochlear implant, including the microphone, connecting cables, speech processor, and transmitter coil, detect the sound and convey the information to the receiving coil and electrode array. These internal components are surgically placed within the mastoid bone and cochlea, respectively, and provide direct electrical stimulation of the auditory nerve (Zwolan, 2002), as can be seen in Figure 7b.

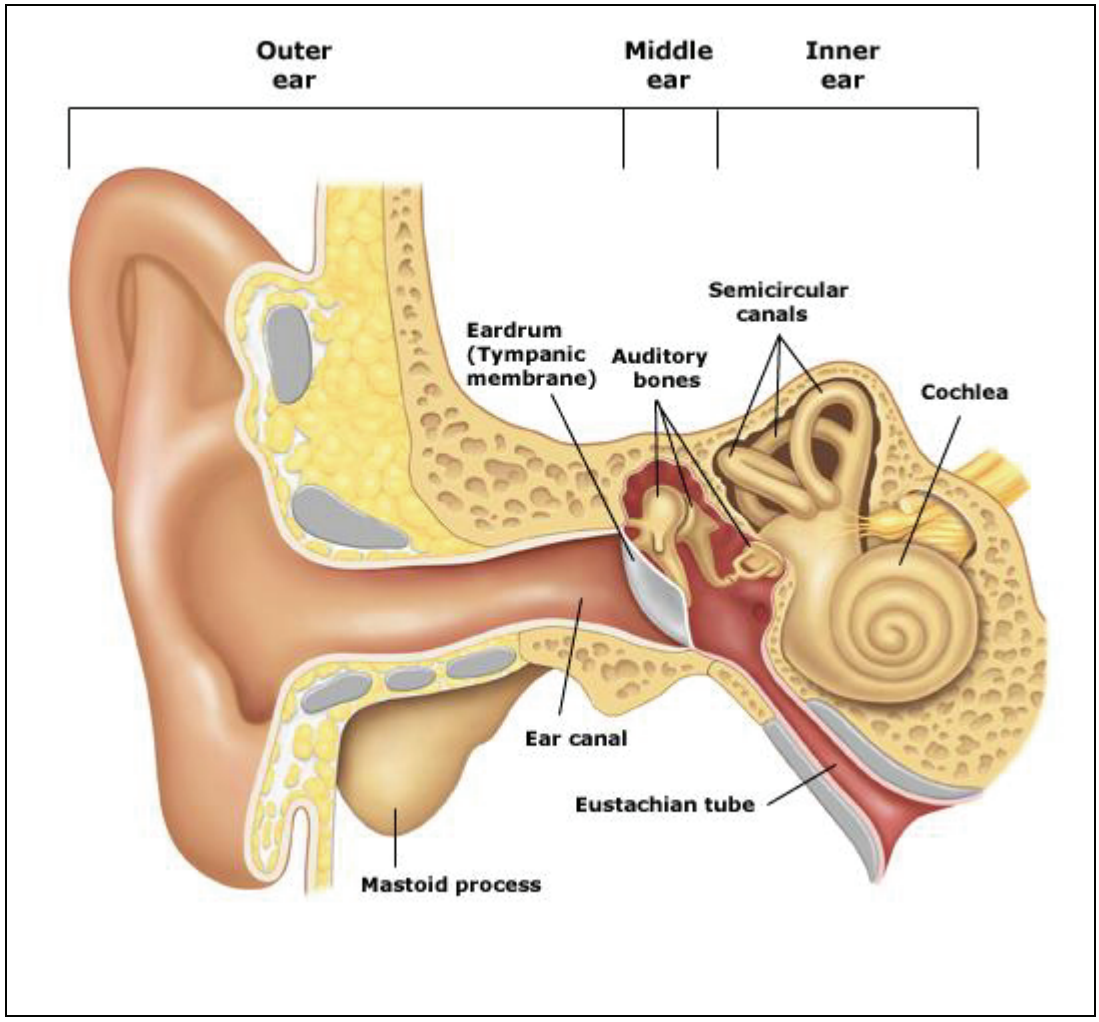


Figure 7a: The peripheral auditory system is comprised of three main sections. The outer ear includes the pinna, ear canal, and the tympanic membrane. The middle ear is made up of the three auditory bones, including the malleus, incus, and stapes, and the Eustacian tube. The final section, the inner ear, is comprised of the cochlea and the semi-circular canals.

The central auditory pathway of a child with a cochlear implant is, therefore, stimulated in a different manner to those children with normal hearing or with hearing aids. However, once the auditory nerve is stimulated by the electrode array, the auditory nervous system should presumably proceed as normal (Zwolan, 2002).

A number of studies have now been published illustrating the use of CAEPs in children and adults with cochlear implants (Eggermont, Ponton, Don, Waring, & Kwong, 1997; Kelly, Purdy, & Thorne, 2005; Ponton, Don, Eggermont, Waring, Kwong et al., 1996; Sharma, Dorman, & Kral, 2005). Ponton and colleagues (1996) investigated the maturation of CAEPs in six children who received their cochlear implant between 18 months and six years of age, with the average age of implantation being 4.5 years. They found that the CAEPs, and in particular, the peak latency of P1, appeared to mature at the same rate as in children with normal hearing. However, the maturation seemed to be delayed by the corresponding length of auditory deprivation (in this case, around 4.5 years; Ponton, Don, Eggermont, Waring, & Masuda, 1996).

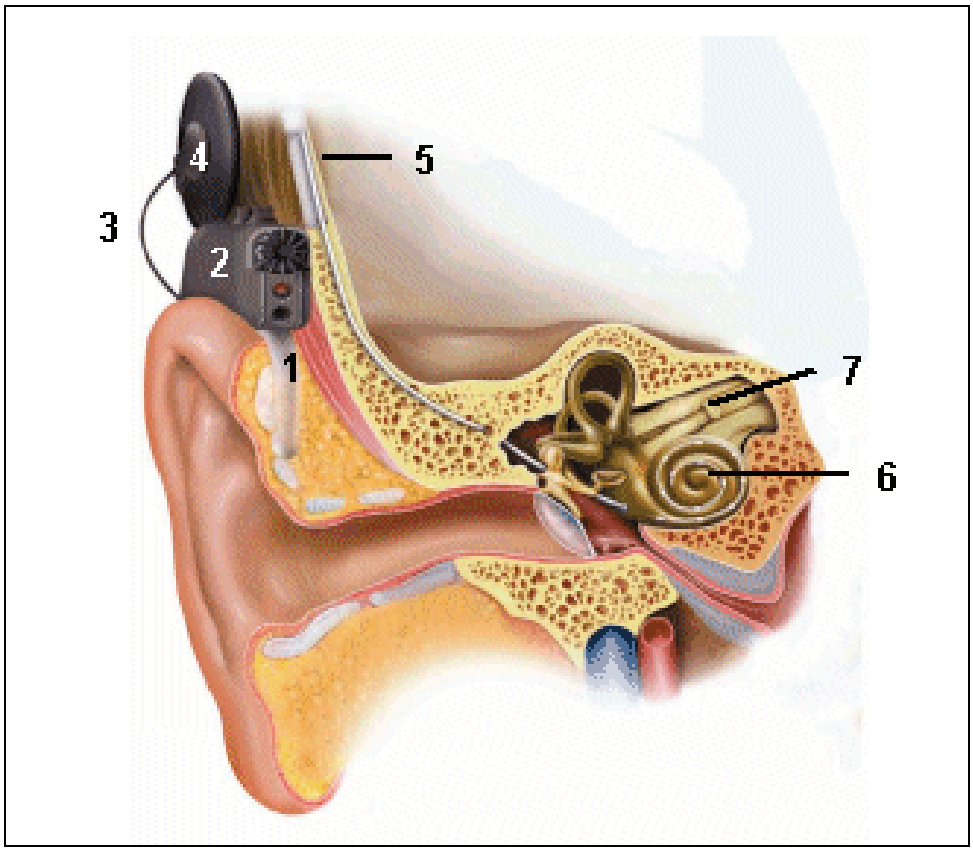


Figure 7b: Sound enters the microphone (1) and is passed on to the speech processor (2) which analyses and translates the signal into an electronic code. The code passes along the connecting cable (3) to the transmitting coil (4) where it traverses through the skin to the receiver coil (5) using frequency modulated radio waves. The electrode array (6) within the cochlea then provides electrical stimulation based upon the characteristics of the transmitted code, resulting in electrical impulses to progress from the auditory nerve (7) to the auditory cortex via the central auditory pathway. Figure from Gross, 2003.

These findings were further supported in two additional studies by Ponton and colleagues (1996, 1997), where both studies investigated 12 children who received their cochlear implant between five months and eight years of age. In both these studies they demonstrated that once stimulation is received via the

cochlear implant, the central auditory system appears to continue to develop at the same rate as in children with normal hearing, but the maturation is delayed by the length of auditory deprivation (Eggermont et al., 1997; Ponton, Don, Eggermont, Waring, Kwong et al., 1996). Therefore, these studies imply that, even after periods of auditory deprivation, the central auditory nervous system still has the ability to continue to develop once appropriate stimulation is received, such as that achieved through a cochlear implant (Eggermont et al., 1997; Ponton, Don, Eggermont, Waring, Kwong et al., 1996).

1.5 A Sensitive Period for Central Auditory Nervous System Development

Sharma and colleagues (2002) further investigated the prospect of a sensitive period for the development of the central auditory system in children. They measured CAEPs in 104 children with cochlear implants and compared the waveforms to evoked responses measured from 136 children with normal hearing. The children with cochlear implants were divided into three groups, based on their age at implantation: early (before 3.5 years); mid (3.5-6.5 years); and late (after 7 years) (Sharma, Dorman et al., 2002b). They found that the majority of children who were implanted at an early age had P1 latencies within the normal range appropriate for their age. In contrast, those children who received their cochlear implant after seven years of age exhibited P1 latencies delayed by approximately 100 ms when compared to the normal range for their chronological age (Sharma, Dorman et al., 2002b). The difference between these two groups clearly illustrates the existence of a sensitive period up to 3.5 years of age. If

appropriate amplification is provided during this sensitive period, the auditory system is able to recover from deprivation (Sharma, Dorman et al., 2002b). Children who received a cochlear implant between these two extremes presented with inconsistent outcomes, indicating that there are additional factors which influence the plasticity of the central auditory system (Sharma, Dorman et al., 2002b).

Subsequent studies by Sharma and colleagues have further supported the presence of this sensitive period for the auditory system, (Sharma, Dorman, Spahr, & Todd, 2002; Sharma, Dorman, & Spahr, 2002a). In the first study, they investigated 22 children who had received cochlear implants prior to 3.5 years of age and found that the P1 latencies were normal within eight months of implant use when compared to the normal range for their chronological age (Sharma, Dorman et al., 2002a). In the second study, they investigated 18 children who had received cochlear implants prior to 3.5 years of age and compared their CAEP responses with age-matched children with normal hearing. They found that the latency of P1 was age-appropriate after only six months of implant use (Sharma, Dorman, Spahr et al., 2002). Both these studies provide evidence of a minimally degenerate and/or highly plastic central auditory system which is able to overcome a minimal period of auditory deprivation (Sharma, Dorman, Spahr et al., 2002; Sharma, Dorman et al., 2002a). Furthermore, both studies indicate that the functioning of the central auditory system can be determined through the use of P1 latency measures (Sharma, Dorman, Spahr et al., 2002; Sharma, Dorman et al., 2002a).

1.6 Early versus Late Cochlear Implantation

The morphology and latency of CAEPs in early and late implanted children have been further investigated (Sharma, Dorman et al., 2005). They found that the CAEPs of all children who are congenitally deaf are dominated by a large negativity that precedes P1, as shown in Panel A of Figure 8. The amplitude of this negativity decreases after stimulation is received from the cochlear implant (Sharma, Dorman et al., 2005). It was suggested that this large negativity was similar to the "long-latency negative potential" seen in preterm infants before 25 weeks post-conception, indicating that this may reflect the lack of stimulation in the auditory system (Sharma, Dorman et al., 2005).

Furthermore, the decreases seen in the latency of P1 were larger than children implanted after 7 years of age, and continued to occur after the first month of stimulation, for the children who received their cochlear implant before 3.5 years of age, reaching normal limits within six to eight months (Sharma, Dorman et al., 2005). In contrast, the CAEP morphology of children who received implants after seven years of age remained atypical until 12-18 months of implant use, as displayed in Panel B of Figure 8 (Sharma, Dorman et al., 2005).

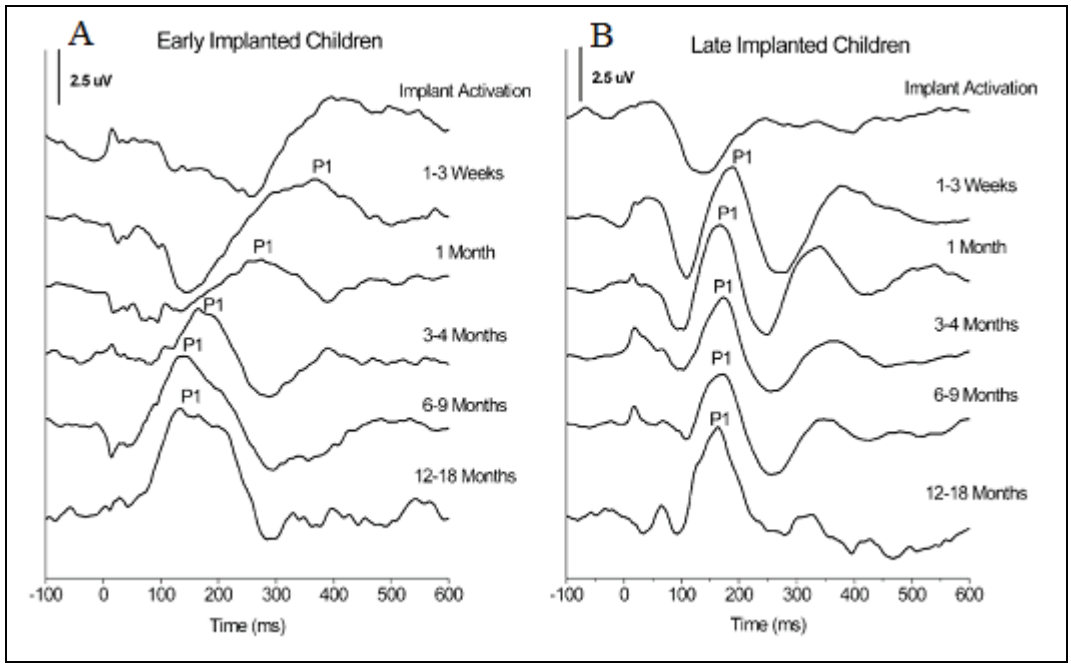


Figure 8: Grand average CAEPs for children who received cochlear implants prior to 3.5 years (Panel A – Early Implanted) and after 7 years (Panel B – Late Implanted). P1 latency decreases in the early implanted children but remains stable in the late implanted children. A large negativity precedes all profoundly deaf children's waveforms prior to receiving any stimulation.

CAEP measurements could therefore be clinically useful to confirm the function of the auditory pathways. Such information would be clinically valuable for determining whether appropriate stimulation was being provided by a hearing aid or cochlear implant, particularly in hard-to-test populations, including young infants. Sharma and colleagues (2005) investigated three children who all received intervention prior to 3.5 years of age. All children had CAEPs recorded before and after a hearing aid trial. In one child, the P1 latency decreased to normal limits within five months of use, indicating the child was receiving adequate amplification from the hearing aid, as seen in Figure 9 (Sharma, Martin

et al., 2005). However, for the other two children, no changes in CAEP morphology were seen. Cochlear implants were therefore provided, after which the P1 latency returned to normal limits within three to six months after use, as displayed in Figures 10 and 11. This indicated that appropriate amplification was only provided after implantation, and thus provides objective support for the use of cochlear implants in these children (Sharma, Martin et al., 2005).

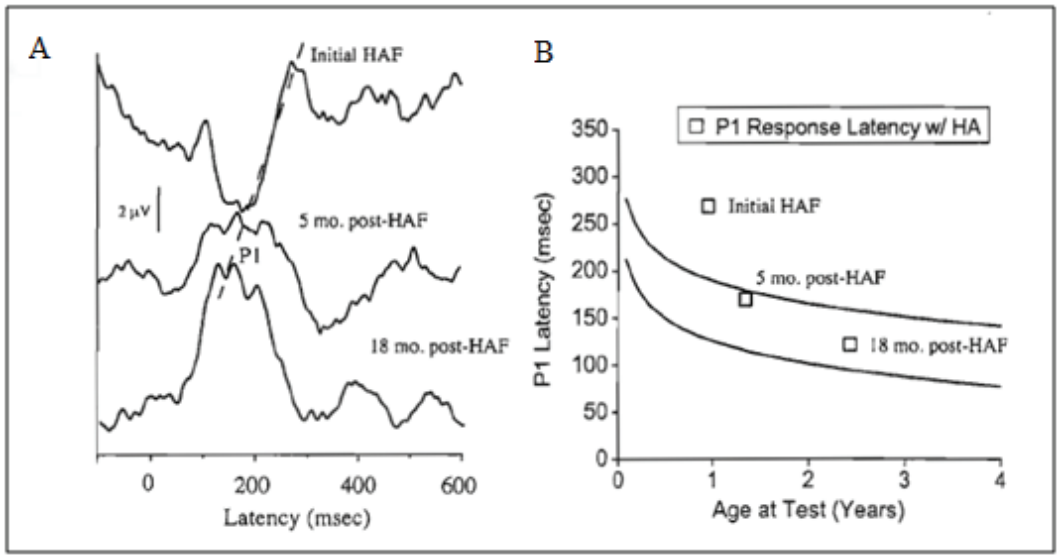


Figure 9: Panel A shows the cortical auditory evoked potentials of a child at their initial hearing aid fitting (HAF), and at 5 months and 18 months post-HAF, clearly demonstrating the decrease in P1 latency. Panel B shows P1 latency as a function of age against the 95% confidence intervals for normal hearing children. This illustrates that within 5 months post-HAF the P1 latency decreased to within the normal developmental range. Figures redrawn from Sharma, Martin et al. (2005).

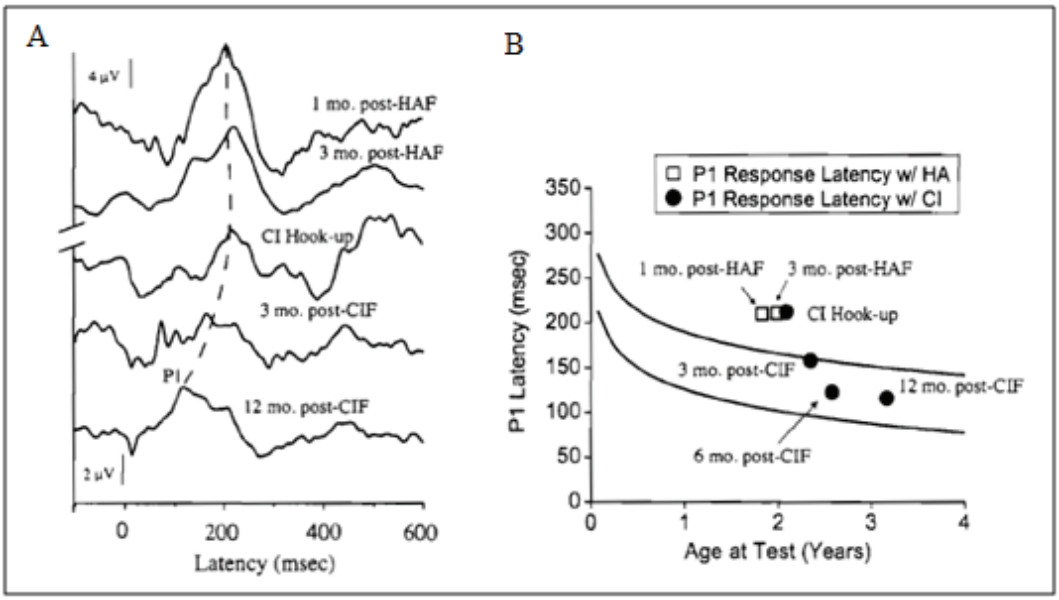


Figure 10: Panel A shows the cortical auditory evoked potentials of a child at their initial hearing aid fitting (HAF), and at 3 months post-HAF, then at their cochlear implant (CI) hook-up, and at 3 months and 12 months post-CIF (cochlear implant fitting). This clearly demonstrates the decrease in P1 latency only following placement of the cochlear implant. Panel B shows P1 latency as a function of age against the 95% confidence intervals for normal hearing children. This illustrates that within 3 months post-CIF the P1 latency decreased to within the normal developmental range. Figures redrawn from Sharma, Martin et al. (2005).

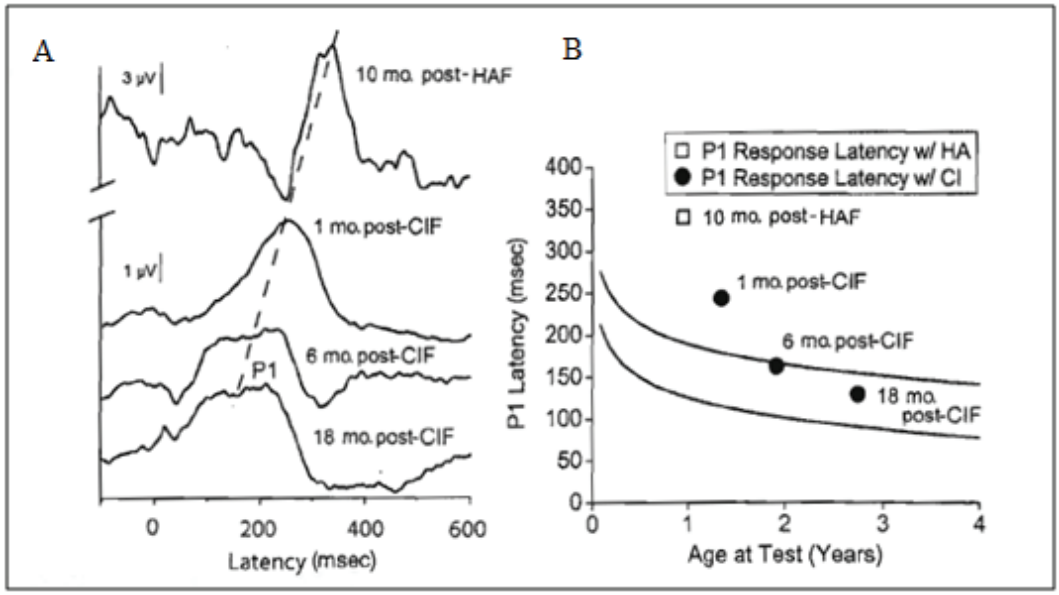


Figure 11: Panel A shows the cortical auditory evoked potentials of a child at 10 months post-HAF (hearing aid fitting), then at 1 month, 6 months, and 18 months post-CIF (cochlear implant fitting). This clearly demonstrates the decrease in P1 latency only following placement of the cochlear implant. Panel B shows P1 latency as a function of age against the 95% confidence intervals for normal hearing children. This illustrates that within 6 months post-CIF the P1 latency decreased to within the normal developmental range. Figures redrawn from Sharma, Martin et al. (2005).

1.7 Conflicting Maturation Data and Aims of the Study

Despite these studies supporting the use of CAEPs in children with cochlear implants, the maturation of evoked potentials is still not well understood. In particular, there is conflicting evidence from the two major researchers in this field. Ponton and colleagues' data demonstrate a delay in the maturation of CAEP responses equal to the time that the child spent without adequate auditory stimulation (Ponton, Don, Eggermont, Waring, Kwong et al., 1996; Ponton, Don,

Eggermont, Waring, & Masuda, 1996). This is in direct contrast with Sharma and colleagues' data that illustrates that CAEP responses develop to the point of becoming age-appropriate once appropriate amplification is provided (Sharma, Dorman et al., 2005; Sharma, Dorman et al., 2002b).

The aim of the present study was, therefore, to investigate the maturation of CAEPs in children who received their cochlear implant prior to 3.5 years of age compared to children with normal hearing who are matched for time-in-sound, as well as to children with normal hearing who are matched for chronological age. It was hoped that this would provide clear evidence as to whether CAEPs in children with cochlear implants continue to mature normally but at a delayed rate (as proposed by Ponton and colleagues) or whether the evoked responses become age-appropriate after a period of time (as proposed by Sharma and colleagues). Furthermore, in order to assess whether the length of time in sound was the critical factor, or whether it was stimulation from a cochlear implant or hearing aids, a direct comparison between children using a cochlear implant versus hearing aids was made.

If CAEPs in children with cochlear implants were found to continue to mature at a normal rate, albeit delayed by the time spent with inadequate auditory stimulation, then this study would support the importance of providing appropriate auditory stimulation as early as possible. However, if the data supported the notion that the CAEPs are able to recover so long as appropriate stimulation is provided prior to

3.5 years of age, then this indicates the need for early intervention to occur during this sensitive period in order to maximise the benefits of the hearing instrument. Therefore, it was hoped that this information would help us understand the maturation of CAEPs in children who receive cochlear implants prior to 3.5 years of age, and assist in establishing ways in which these objective measures could be used to support appropriate implantation and ensure adequate stimulation is being received.

It was hypothesised this study would find that children who received a cochlear implant prior to 3.5 years of age would have CAEP waveforms equivalent to their chronologically age-matched, normal hearing peers. Furthermore, we hypothesised that the evoked responses in the child with a hearing impairment who received a hearing aid before 3.5 years of age would also be identical to their chronologically age-matched peers who either had a cochlear implant or normal hearing, as adequate stimulation was being provided.

Chapter 2

Method

2.1 Participants

The participants included five children with cochlear implants who received their cochlear implant prior to 3.5 years of age. The child's experience with the cochlear implant, or "time-in-sound", varied from participant to participant. The demographic information for the participants (all drawn from the Southern Cochlear Implant Program) can be seen in Table 1.

Table 1: Demographic Information for Cochlear Implant Children

The demographic information for the five children with cochlear implants (CI) who received their cochlear implant prior to 3.5 years of age.

Participant	Chronological Age (y, m)	Age at Implant (y, m)	Time-in-Sound (y, m)	Ear	Implant	Array
CI 1	5y, 3m	1y, 9m	3y, 6m	left	Nucleus 24	CI24RE(CS)
CI 2	8y, 3m	3y, 1m	5y, 1m	right	Nucleus 24	CI24RE(CS)
CI 3	8y, 3m	2y, 1m	6y, 1m	right	Nucleus 24	CI24RE(CS)
CI 4	10y, 3m	3y, 2m	7y, 1m	right	Nucleus 24	CI24RE(CS)
CI 5	12y, 5m	3y, 0m	9y, 4m	right	Nucleus 24	CI224M

y = years; m = months

CAEPs were also recorded from children with normal hearing, including five children who were matched based on chronological age, and five children who were matched based on "time-in-sound" (for example, a five year old child who had worn a cochlear implant for two years was matched with a five year old child with normal hearing, as well as with a two year old child with normal hearing).

These children were drawn from local schools, and had their hearing screened to ensure they had normal hearing at 20 dB HL across the frequencies 500-4000 Hz.

Finally, a 10 year old child who was fitted with hearing aids prior to 3.5 years of age was tested and compared with a 10 year old child who also received a cochlear implant prior to 3.5 years of age (CI4). The child with hearing aids had a congenital moderate sloping to severe sensorineural hearing loss bilaterally. The participant was drawn from the existing caseload of a local Advisor of Deaf Children employed by the Ministry of Education Special Education.

The study was approved by both the Upper South A Regional Health and Disability Ethics and the University of Canterbury Human Ethics Committees, as shown in Appendices 1 and 2, respectively. All children and their families were provided with an Information Sheet, a copy of which can be seen in Appendix 3. A copy of the Consent Form, which was signed by the child's parent, is shown in Appendix 4.

2.2 Stimulus

Two stimuli were used to evoke the CAEPs: a recorded consonant-vowel speech stimulus /ba/, and a 193 Hz pure-tone. Although any auditory stimulus can be used to evoke a CAEP response, speech stimuli frequently elicit a more robust waveform (Wunderlich et al., 2006). The specific consonant-vowel syllable /ba/ was selected for a number of reasons. Firstly, the high contrast between the

consonant and the following vowel makes it a strong stimulus. Furthermore, a voice recording was completed to generate the syllable to allow for a more natural sounding syllable and again increasing the strength of the stimulus. Finally, /ba/ was chosen so that comparisons could be made between the current study and the numerous studies reported by Sharma and colleagues in which this stimulus has been used (Sharma, Dorman, Spahr et al., 2002; Sharma, Dorman et al., 2005; Sharma, Dorman et al., 2002b; Sharma et al., 1997; Sharma, Martin et al., 2005). However, it is important to note the /ba/ syllable used by Sharma and colleagues was synthesised, whereas the speech stimulus used in the current study was recorded, resulting in differences between the formant frequencies. The speech syllable was recorded by a 27 year old female using a condenser microphone (AKG C420) which was connected to a microphone amplifier (Eurorack MX602A, Behringer). The 193 Hz pure-tone stimulus was used to verify these results as this was the frequency of the first formant of the recorded consonant-vowel speech stimulus used in the current study.

Both stimuli were 90 ms in duration, with a 10 ms rise and fall time to prevent any “spectral splatter” arising from the rapid onset or offset of the waveform. The waveforms for the speech stimulus and the pure-tone are shown in Figure 12.

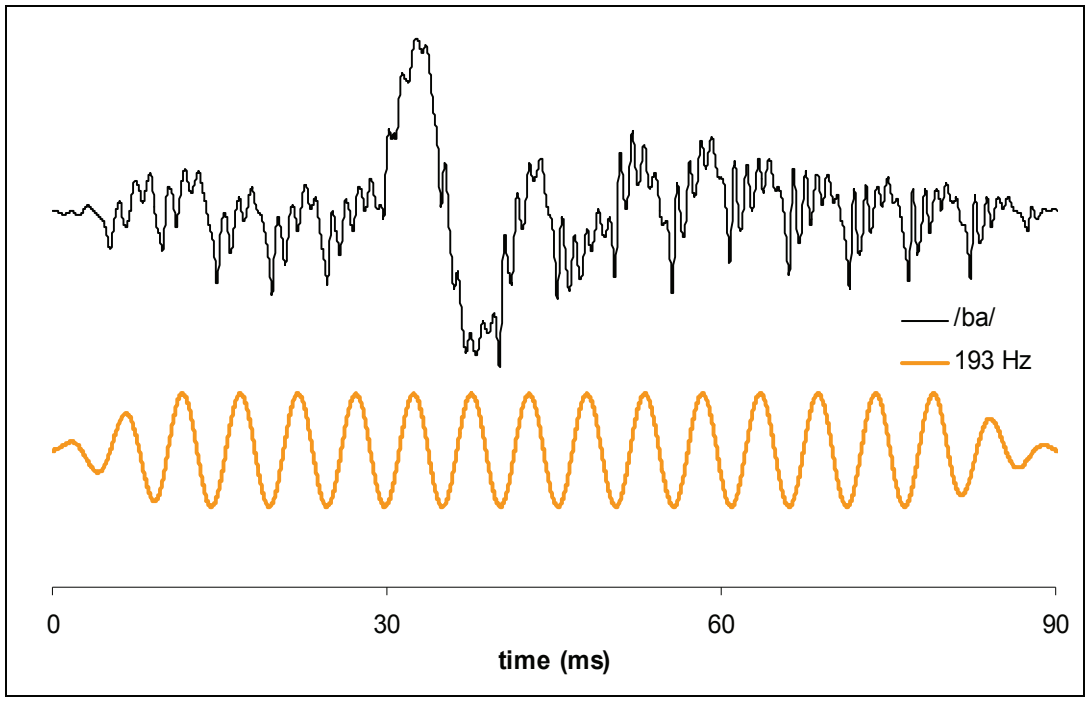


Figure 12: The two stimulus waveforms, recorded /ba/ speech stimulus (top) and 193 Hz pure-tone (bottom) are displayed. Both stimuli were 90 ms in duration with a 10 ms rise and fall time.

An interstimulus interval (ISI) of 610 ms was used, as it has been shown that a slower stimulation rate results in more robust CAEP waveforms in immature auditory nervous systems (Gilley, Sharma, Dorman, & Martin, 2005). However, any ISI longer than this tends to result in extended session times without adding additional information of significant value (Gilley et al., 2005).

2.3 Stimulus Presentation

The stimuli were presented at 70 dB SPL (as measured at the child's head) which approximates normal conversational level. It was confirmed with each child that this was at a loud but comfortable listening level. Presentation was via a loudspeaker placed at a 45 degree angle to the side of the cochlear implant. The speaker was then positioned on the same side for the matched children with normal hearing. For the child with hearing aids, the speaker was placed on the side of their better hearing ear, to most closely approximate the set-up of the children with cochlear implants.

2.4 Set-Up

The child sat on a comfortable chair in a sound-attenuated room at the University of Canterbury Speech and Hearing Clinic, shown in Figure 13. Those children with cochlear implants or hearing aids ensured their hearing instrument was set to their normal settings and any noise reduction functions were deactivated. This was to ensure that the hearing instruments did not alter the acoustic stimuli, such as determining the sound to be noise and then damping it as a result. Children with cochlear implants switched off any hearing instruments worn on the contralateral ear. However, residual hearing was not blocked out. The child was allowed to watch a children's DVD of their own choice. McArthur, Bishop, and Proudfoot (2003) showed that the presence of low-level video sound did not significantly impact on the P1-N1-P2 waveform in adults. The volume of the

DVD was, therefore, kept below 40 dB SPL, which helped to keep the child engaged without interfering with the stimulus.

Figure 13: A photograph of the testing room. A child is seated on a comfortable chair in front of the television where a children's movie is playing and the speaker which presents the stimuli is placed at a 45 degree angle.

2.5 Recording

Three electrodes were placed on the child's head to record the CAEPs. The active electrode was placed at Cz. The reference electrode was placed on the mastoid of the contralateral ear. The ground electrode was placed on the cheek.

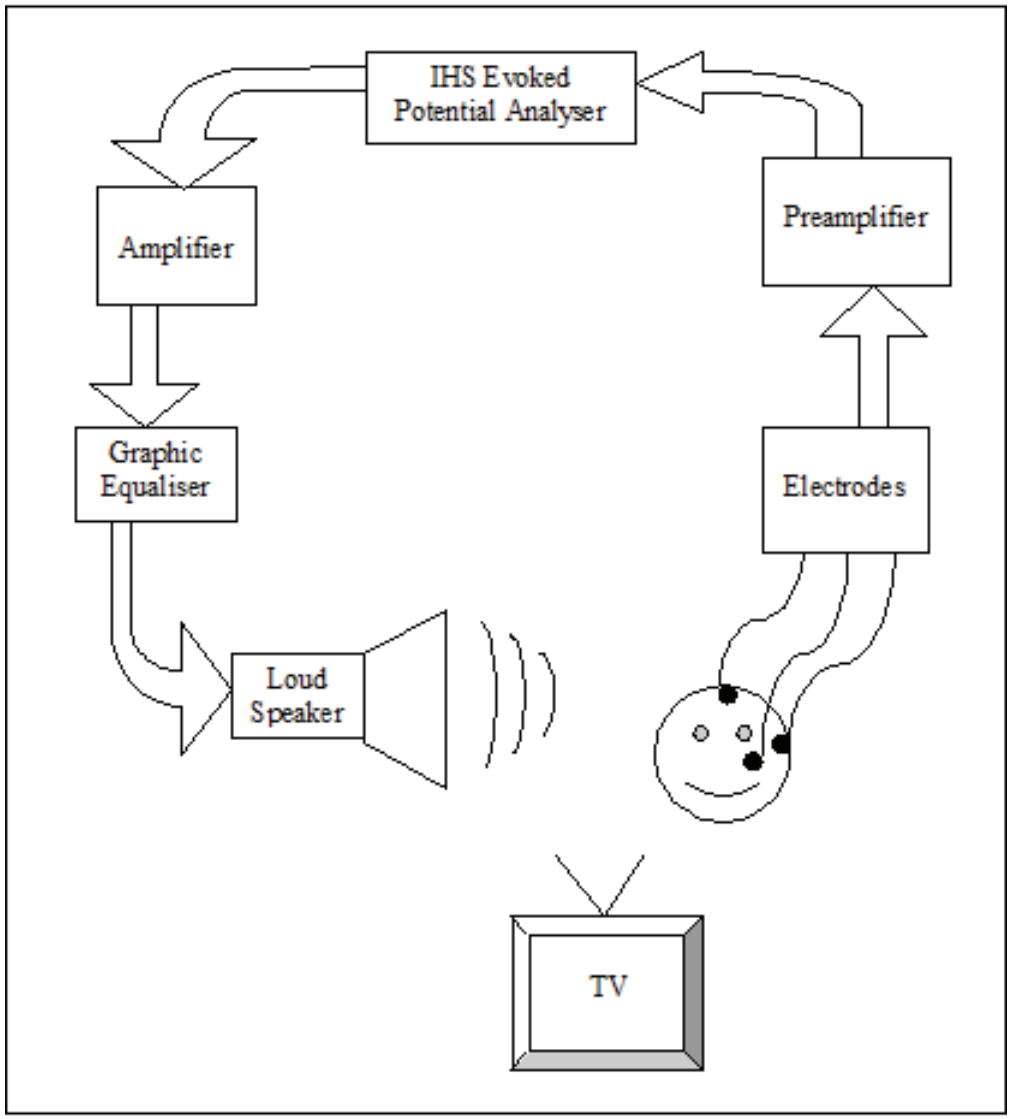


Figure 14: A diagram demonstrating the set-up of the room and computer equipment. The participant is seated in front of the television (TV) which is playing a children's movie. Three electrodes were placed at Cz, the mastoid, and the cheek. The electrodes were then connected to the preamplifier before passing on to the Intelligent Hearing Systems (IHS) evoked potential analyser, which both analysed the incoming data as well as generating the auditory stimulus. This stimulus travelled via the amplifier and graphic equaliser before being presented through the loud speaker and detected by the participant.

An Intelligent Hearing Systems (IHS) Evoked Potential Analyser was used to record the responses, as shown in Figure 14. Responses were sampled at a 1000 Hz frequency rate and analog filtered from 0.1 to 100 Hz, with at least 1000 responses recorded per stimulus for each child. Each test session lasted no longer than 60 minutes, including the electrode application and CAEP recording.

2.6 Data Analysis

The raw responses recorded were collected in four recordings of 256 responses elicited by the 193 Hz pure-tone and the /ba/ speech stimulus, which are displayed in Appendix 5. These waveforms were visually judged as being replicable and then added together to create one averaged waveform per stimulus for each child. The four peaks were identified and peak latency and amplitude measures were made for each of the four waveforms. These were then averaged together to get means and standard deviations for both latency and amplitude. This was completed for all four peaks.

Peak latency measures were made at the most positive or negative point and peak amplitude measures were made from baseline to peak. Previous research found no difference in results when using peak amplitude or area as the measurement of comparison. This was also true with peak and onset latency measurements (Wunderlich et al., 2006).

One-way repeated measures analysis of variance (ANOVA) tests were used to determine whether there were significant differences in the latency and amplitude measures of the various peaks between the different groups of children. The significance level was set at 0.05.

Chapter 3

Results

3.1 Effect of Stimulation on Peak Latencies and Amplitudes

Two stimuli, a pure-tone and a recorded consonant-vowel, were used to elicit CAEPs in children with hearing instruments and normal hearing. The 193 Hz pure-tone was selected as this was the fundamental frequency of the /ba/ speech stimuli. Therefore, paired 2-tailed t-tests were used to investigate whether there were any significant differences between the latency and amplitude measurements recorded by the two stimuli for each individual child.

The statistical data that highlights any significant differences between the waveforms elicited by the 193 Hz pure-tone and the /ba/ speech stimuli are shown in Table 2. This information is also detailed in the following Sections 3.1.1 to 3.1.4.

193 Hz vs /ba/

Participant	P1 latency	P1 amplitude	N1 latency	N1 amplitude	P2 latency	P2 amplitude	N2 latency	N2 amplitude
CI 1	Not significant	Sig (p=0.036)	Not significant	Not significant	Not significant	Not significant	Sig (p=0.009)	Sig (p=0.002)
CI 2	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant
CI 3	Not significant	Not significant	Not significant	Sig (p=0.010)	Not significant	Sig (p=0.017)	Not significant	Not significant
CI 4	Not significant	Not significant	Not significant	Sig (p=0.023)	Not significant	Sig (p=0.008)	Not significant	Sig (p=0.017)
CI 5	Sig (p=0.019)	Not significant	Not significant	Not significant	Sig (p=0.018)	Sig (p=0.010)	Sig (p=0.012)	Not significant
NH 1	Not significant	Not significant	Not significant	Not significant	Sig (p=0.001)	Not significant	Sig (p=0.023)	Not significant
NH 2	Not significant	Sig (p=0.020)	Not significant	Sig (p=0.008)	Sig (p=0.021)	Sig (p=0.023)	Sig (p=0.033)	Sig (p=0.004)
NH 3	Sig (p=0.015)	Sig (p=0.024)	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant
NH 4	Sig (p=0.002)	Sig (p=0.033)	Not significant	Not significant	Not significant	Not significant	Not significant	Sig (p=0.004)
NH 5	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Sig (p=0.043)
TiS 1	Not significant	Not significant	Not significant	Not significant	Not significant	Sig (p=0.008)	Not significant	Not significant
TiS 2	Not significant	Sig (p=0.047)	Not significant	Not significant	Sig (p=0.022)	Not significant	Not significant	Sig (p= 0.010)
TiS 3	Not significant	Sig (p=0.019)	Not significant	Not significant	Not significant	Sig (p=0.007)	Not significant	Sig (p=0.014)
TiS 4	Sig (p=0.011)	Sig (p=0.007)	Not significant	Not significant	Sig (p=0.010)	Sig (p=0.000)	Not significant	Sig (p=0.011)
TiS 5	Not significant	Sig (p=0.038)	Not significant	Not significant	Not significant	Not significant	Not significant	Sig (p=0.034)
HA 4	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant	Not significant

Table 2: Statistical data for 193 Hz versus /ba/

The statistically significant data (sig) for the latency and amplitude measurements between the waveforms evoked by the 193 Hz pure-tone and the recorded /ba/ speech stimuli. Data is shown for children with cochlear implants (CI), chronologically age-matched normal hearing children (NH), and time-in-sound age-matched normal-hearing children (TiS), and a chronologically age-matched child with hearing aids (HA). The significance level was set at 0.05.

3.1.1 P1 Latency and Amplitude

The P1 latency and amplitude measurements recorded in response to the 193 Hz pure-tone and the /ba/ recorded consonant-vowel speech stimulus for all children are displayed in Table 3.

The difference in the P1 latency measurements in response to the 193 Hz pure-tone and to the recorded /ba/ stimulus was not found to be significantly different for the child with hearing aids [($p<0.05$), $t(3) = -0.14$, $p = 0.899$] or for four of the children with cochlear implants [CI 1: ($p<0.05$), $t(3) = 0.14$, $p = 0.895$; CI 2: ($p<0.05$), $t(3) = 0.91$, $p = 0.428$; CI 3: ($p<0.05$), $t(3) = -1.45$, $p = 0.243$; CI 4: ($p<0.05$), $t(3) = 0.35$, $p = 0.750$]. However, CI 5 was found to have a significant difference, [($p<0.05$), $t(3) = 4.63$, $p = 0.019$], in the P1 latency measurements recorded in response to these two stimuli.

Similar results were found for the children with normal hearing with three of the children matched for chronological age [NH 1: ($p<0.05$), $t(3) = -2.89$, $p = 0.063$; NH 2: ($p<0.05$), $t(3) = -0.71$, $p = 0.530$; NH 5: ($p<0.05$), $t(3) = -0.62$, $p = 0.581$] and four of the children matched for "time-in-sound" [TiS 1: ($p<0.05$), $t(3) = -1.6$, $p = 0.208$; TiS 2: ($p<0.05$), $t(3) = -0.73$, $p = 0.521$; TiS 3: ($p<0.05$), $t(4) = 0.19$, $p = 0.857$; TiS 5: ($p<0.05$), $t(3) = 2.42$, $p = 0.094$] showing no significant difference in their P1 latency measurements recorded in response to either stimuli. The P1 latencies of NH 3 [($p<0.05$), $t(3) = -5.07$, $p = 0.015$], NH 4 [($p<0.05$), $t(3) = -10.5$,

p = 0.002], and TiS 4 [($p<0.05$), $t(4) = 5.56$, $p = 0.011$], were statistically significant when different stimuli were used to evoke the CAEPs.

Most children with hearing instruments had no statistically significant differences [CI 2: ($p<0.05$), $t(3) = 2.63$, $p = 0.078$; CI 3: ($p<0.05$), $t(3) = 5.39$, $p = 0.013$; CI 4: ($p<0.05$), $t(3) = -3.05$, $p = 0.055$; CI 5: ($p<0.05$), $t(3) = -1.02$, $p = 0.384$; HA 4: ($p<0.05$), $t(3) = -1.68$, $p = 0.192$] in their P1 amplitude measurements when elicited using either the pure-tone or speech stimulus. CI 1 did show a statistically significant difference in P1 amplitude [($p<0.05$), $t(3) = -3.64$, $p = 0.036$].

Table 3: P1 Peak Latency and Amplitude Measurements

The P1 latency and amplitude measurements recorded in response to a 193 Hz pure-tone and a recorded /ba/ speech stimulus, presented at 70 dB SPL, for children with cochlear implants (CI), chronologically age matched normal hearing children (NH), time-in-sound age matched normal hearing children (TiS), and a chronologically age matched child with hearing aids (HA). Means and standard deviations are shown (n = 4 replicates of 256 averages).

Participant	193 Hz - P1 Latency (ms)	193 Hz - P1 Amplitude (μV)	/ba/ - P1 Latency (ms)	/ba/ - P1 Amplitude (μV)
CI 1	115 ± 4.7	4.0 ± 1.3	114.8 ± 4.6	7.4 ± 1.2
CI 2	92.8 ± 3.2	4.1 ± 1.7	95.7 ± 4.9	2.9 ± 1.0
CI 3	83.3 ± 6.7	4.4 ± 0.6	89.3 ± 2.5	3.7 ± 0.5
CI 4	86.8 ± 4.8	4.6 ± 0.8	85 ± 8.0	8.5 ± 2.4
CI 5	95.8 ± 5.6	1.8 ± 1.0	85 ± 4.2	2.4 ± 1.0
NH 1	88 ± 1.4	2.7 ± 1.8	97.3 ± 7.3	3.0 ± 0.9
NH 2	90.6 ± 11.4	0.8 ± 0.4	90.3 ± 1.5	3.4 ± 1.1
NH 3	76.2 ± 16.0	0.3 ± 0.7	103.8 ± 5.2	3.1 ± 1.5
NH 4	60.3 ± 3.4	1.5 ± 0.2	97.3 ± 5.3	3.1 ± 1.0
NH 5	77.8 ± 20.5	0.5 ± 0.3	84 ± 11.6	1.4 ± 0.9
TiS 1	88.3 ± 2.6	2.9 ± 0.8	107 ± 22.8	2.9 ± 0.7
TiS 2	75.3 ± 20.0	0.4 ± 0.6	83.3 ± 10.3	2.7 ± 1.1
TiS 3	95 ± 9.7	2.0 ± 0.5	93.8 ± 6.2	3.8 ± 0.7
TiS 4	100.5 ± 4.7	1.9 ± 0.8	86.5 ± 2.4	4.6 ± 0.2
TiS 5	95 ± 2.0	0.4 ± 0.6	83.8 ± 9.4	1.9 ± 1.0
HA 4	90.5 ± 9.6	3.0 ± 0.7	91.3 ± 7.2	3.5 ± 0.7

ms = milliseconds; μV = microvolts

The P1 amplitude measurements of only three children with normal hearing were found to have no statistical significance [NH 1: ($p<0.05$), $t(3) = -0.44$, $p = 0.689$; NH 5: ($p<0.05$), $t(3) = -2.55$, $p = 0.084$; TiS 1: ($p<0.05$), $t(3) = 0.452$, $p = 0.967$]. Significant differences were found in the P1 amplitudes recorded for the other three chronologically age-matched normal hearing children [NH 2: ($p<0.05$), $t(3) = -4.54$, $p = 0.02$; NH 3: ($p<0.05$), $t(3) = -4.26$, $p = 0.024$; NH 4: ($p<0.05$), $t(3) = -3.74$, $p = 0.033$] and four of the normal hearing children matched for "time-in-sound" [TiS 2: ($p<0.05$), $t(3) = -3.26$, $p = 0.047$; TiS 3: ($p<0.05$), $t(4) = -3.80$, $p = 0.019$; TiS 4: ($p<0.05$), $t(3) = -6.53$, $p = 0.007$; TiS 5: ($p<0.05$), $t(3) = -3.54$, $p = 0.038$].

3.1.2 N1 Latency and Amplitude

The N1 latency and amplitude means and standard deviations recorded in response to the pure-tone and the recorded speech stimulus are shown in Table 4 for children with cochlear implants, normal hearing, and the child with hearing aids.

Table 4: N1 Peak Latency and Amplitude Measurements

The N1 latency and amplitude measurements recorded in response to a 193 Hz pure-tone and a recorded /ba/ speech stimulus, presented at 70 dB SPL, for children with cochlear implants (CI), chronologically age matched normal hearing children (NH), time-in-sound age matched normal hearing children (TiS), and a chronologically age matched child with hearing aids (HA). Means and standard deviations are shown (n = 4 replicates of 256 averages).

Participant	193 Hz - N1 Latency (ms)	193 Hz - N1 Amplitude (μV)	/ba/ - N1 Latency (ms)	/ba/ - N1 Amplitude (μV)
CI 1	161.5 ± 6.6	-1.8 ± 0.4	163.8 ± 2.1	-0.5 ± 1.2
CI 2	152.5 ± 11.9	-3.9 ± 0.7	144 ± 26.4	-1.2 ± 2.4
CI 3	148.3 ± 4.6	-5.6 ± 0.6	184.5 ± 25.8	-4.2 ± 0.4
CI 4	152.8 ± 14.5	-5.0 ± 0.9	165.5 ± 16.8	-8.6 ± 1.4
CI 5	157.8 ± 18.5	-2.3 ± 0.3	127 ± 7.6	-2.9 ± 0.3
NH 1	166 ± 13.7	-2.7 ± 0.6	171.3 ± 6.8	-1.9 ± 0.3
NH 2	138 ± 10.4	-1.3 ± 0.6	147.5 ± 5.9	-2.5 ± 0.7
NH 3	136.4 ± 31.2	-2.7 ± 0.7	157.5 ± 19.6	-3.3 ± 1.6
NH 4	139 ± 7.1	-3.0 ± 0.5	149.3 ± 9.1	-3.8 ± 1.2
NH 5	129.5 ± 5.0	-2.1 ± 0.3	132.8 ± 35.1	-1.9 ± 0.6
TiS 1	187 ± 10.7	-2.1 ± 0.9	184.8 ± 29.6	-1.8 ± 1.6
TiS 2	131.3 ± 6.8	-3.1 ± 1.3	126 ± 6.7	-1.8 ± 0.8
TiS 3	169.8 ± 16.6	-3.1 ± 1.1	188.2 ± 35.5	-3.6 ± 1.3
TiS 4	167.5 ± 8.8	-2.9 ± 0.7	157 ± 20.6	-3.0 ± 0.4
TiS 5	139 ± 28.7	-2.4 ± 0.4	134.8 ± 11.8	-2.1 ± 0.4
HA 4	142 ± 7.4	-5.7 ± 0.8	168.8 ± 30.4	-4.2 ± 2.6

ms = milliseconds; μV = microvolts

All of the children with cochlear implants recorded N1 latency measurements with no statistically significant differences [CI 1: ($p<0.05$), $t(3) = -0.82$, $p = 0.473$; CI 2: ($p<0.05$), $t(3) = -0.2$, $p = 0.854$; CI 3: ($p<0.05$), $t(3) = -0.28$, $p = 0.068$; CI 4: ($p<0.05$), $t(3) = 0.91$, $p = 0.431$; CI 5: ($p<0.05$), $t(3) = 2.52$, $p = 0.086$] when elicited by pure-tone and speech stimuli. This was also the case for HA 4 [($p<0.05$), $t(3) = -1.5$, $p = 0.230$].

The N1 latencies of the children with normal hearing matched for chronological age [NH 1: ($p<0.05$), $t(3) = -1.19$, $p = 0.321$; NH 2: ($p<0.05$), $t(3) = -2.33$, $p = 0.102$; NH 3: ($p<0.05$), $t(3) = -2.03$, $p = 0.135$; NH 4: ($p<0.05$), $t(3) = -1.58$, $p = 0.212$; NH 5: ($p<0.05$), $t(3) = -0.2$, $p = 0.853$] and matched for "time-in-sound" [TiS 1: ($p<0.05$), $t(3) = 0.11$, $p = 0.918$; TiS 2: ($p<0.05$), $t(3) = 1.5$, $p = 0.230$; TiS 3: ($p<0.05$), $t(4) = -0.81$, $p = 0.461$; TiS 4: ($p<0.05$), $t(4) = 1.76$, $p = 0.461$; TiS 5: ($p<0.05$), $t(3) = 0.47$, $p = 0.668$] recorded no statistical differences when evoked by either stimuli.

Two of the children with cochlear implants recorded statistically significant differences in the N1 amplitude measurements [CI 3: ($p<0.05$), $t(3) = -5.89$, $p = 0.01$; CI 4: ($p<0.05$), $t(3) = 4.34$, $p = 0.023$]. The other three children with cochlear implants [CI 1: ($p<0.05$), $t(3) = -2.80$, $p = 0.068$; CI 2: ($p<0.05$), $t(3) = -1.76$, $p = 0.176$; CI 5: ($p<0.05$), $t(3) = 2.07$, $p = 0.13$] and the child with hearing aids [HA 4: ($p<0.05$), $t(3) = -1.24$, $p = 0.303$] had no statistical significance between the N1 amplitudes evoked by the two types of stimuli.

No statistically significant differences in N1 amplitude measurements were found for nine out of the ten normal hearing children [NH 1: ($p<0.05$), $t(3) = -2.47$, $p = 0.09$; NH 3: ($p<0.05$), $t(3) = 0.439$, $p = 0.69$; NH 4: ($p<0.05$), $t(3) = 2.07$, $p = 0.131$; NH 5: ($p<0.05$), $t(3) = -0.613$, $p = 0.583$; TiS 1: ($p<0.05$), $t(3) = -0.648$, $p = 0.563$; TiS 2: ($p<0.05$), $t(3) = -1.82$, $p = 0.166$; TiS 3: ($p<0.05$), $t(4) = 1.05$, $p = 0.355$; TiS 4: ($p<0.05$), $t(3) = 0.141$, $p = 0.897$; TiS 5: ($p<0.05$), $t(3) = -2.76$, $p = 0.07$]. One chronologically age-matched child with normal hearing recorded a statistically significant difference in N1 amplitudes [NH 2: ($p<0.05$), $t(3) = 6.45$, $p = 0.008$] when CAEPs were elicited by the pure-tone versus the speech stimulus.

3.1.3 P2 Latency and Amplitude

The means and standard deviations for P2 latency and amplitude measurements from all the children, recorded in response to the 193 Hz pure-tone and the /ba/ recorded consonant-vowel speech stimulus, are displayed in Table 5.

The P2 latency measurements recorded in response to the two different stimuli resulted in no significant difference for all but one of the children with hearing instruments [CI 1: ($p<0.05$), $t(3) = 2.15$, $p = 0.121$; CI 2: ($p<0.05$), $t(3) = 0.92$, $p = 0.932$; CI 3: ($p<0.05$), $t(3) = -0.33$, $p = 0.764$; CI 4: ($p<0.05$), $t(3) = 0.27$, $p = 0.806$; HA 4: ($p<0.05$), $t(3) = -0.32$, $p = 0.767$]. CI 5, was found to have a

statistically significant difference [$p<0.05$), $t(3) = 4.72$, $p = 0.018$] between the two waveforms.

Most of the children with normal hearing showed no statistically significant differences in their P2 latencies [NH 3: ($p<0.05$), $t(3) = -1.5$, $p = 0.231$; NH 4: ($p<0.05$), $t(3) = 2.69$, $p = 0.074$; NH 5: ($p<0.05$), $t(3) = 1.27$, $p = 0.293$; TiS 1: ($p<0.05$), $t(3) = 1.17$, $p = 0.326$; TiS 3: ($p<0.05$), $t(4) = 0.52$, $p = 0.633$; TiS 5: ($p<0.05$), $t(3) = 1.46$, $p = 0.240$]. However, two children matched for chronological age [NH 1: ($p<0.05$), $t(3) = 14.1$, $p = 0.001$; NH 2: ($p<0.05$), $t(3) = 4.44$, $p = 0.021$] and two children matched for "time-in-sound" [TiS 2: ($p<0.05$), $t(3) = 4.38$, $p = 0.022$; TiS 4: ($p<0.05$), $t(4) = 5.86$, $p = 0.010$] recorded statistically significant differences in their P2 latency measurements.

Half of the children with hearing instruments had statistically significant differences in their P2 amplitude measurements [CI 3: ($p<0.05$), $t(3) = 4.84$, $p = 0.017$; CI 4: ($p<0.05$), $t(3) = 6.37$, $p = 0.008$; CI 5: ($p<0.05$), $t(3) = -5.77$, $p = 0.01$]. The other two children with cochlear implants [CI 1: ($p<0.05$), $t(3) = -2.19$, $p = 0.116$; CI 2: ($p<0.05$), $t(3) = -0.384$, $p = 0.727$] and the child with hearing aids [HA 4: ($p<0.05$), $t(3) = -0.470$, $p = 0.671$] recorded no significant differences in the P2 amplitude measurements elicited by the pure-tone and speech stimuli.

Table 5: P2 Peak Latency and Amplitude Measurements

The P2 latency and amplitude measurements recorded in response to a 193 Hz pure-tone and a recorded /ba/ speech stimulus, presented at 70 dB SPL, for children with cochlear implants (CI), chronologically age matched normal hearing children (NH), time-in-sound age matched normal hearing children (TiS), and a chronologically age matched child with hearing aids (HA). Means and standard deviations are shown (n = 4 replicates of 256 averages).

Participant	193 Hz – P2 Latency (ms)	193 Hz – P2 Amplitude (μV)	/ba/ - P2 Latency (ms)	/ba/ - P2 Amplitude (μV)
CI 1	202.5 ± 24.3	0.8 ± 0.7	174.3 ± 8.5	1.5 ± 1.0
CI 2	194.8 ± 9.3	-1.4 ± 0.8	191.8 ± 64.9	-0.8 ± 2.5
CI 3	206 ± 7.0	1.5 ± 0.8	212 ± 35.9	-1.9 ± 1.1
CI 4	202.3 ± 16.8	-0.8 ± 1.4	198.5 ± 22.6	-4.7 ± 1.3
CI 5	251.5 ± 26.4	0.5 ± 0.5	183.8 ± 4.9	2.7 ± 0.7
NH 1	275.5 ± 9.9	0.8 ± 0.9	182.8 ± 3.9	-0.6 ± 0.6
NH 2	239.4 ± 33.6	0.8 ± 0.5	165.8 ± 4.1	-1.2 ± 0.5
NH 3	191.8 ± 57.1	0.5 ± 1.4	183.5 ± 25.5	-0.3 ± 0.7
NH 4	225.3 ± 22.5	1.3 ± 0.8	192 ± 5.5	0.2 ± 0.6
NH 5	183.8 ± 15.7	-0.1 ± 0.5	165 ± 24.5	-0.7 ± 0.8
TiS 1	264.8 ± 21.4	2.4 ± 0.8	225.8 ± 49.5	0.8 ± 1.2
TiS 2	209.8 ± 9.6	3.3 ± 2.1	181 ± 6.2	2.9 ± 0.5
TiS 3	235.6 ± 32.8	2.1 ± 0.8	221.4 ± 53.4	-0.2 ± 1.0
TiS 4	264.5 ± 15.5	0.9 ± 0.3	176.8 ± 27.3	-1.3 ± 0.4
TiS 5	205.8 ± 13.4	0.1 ± 0.4	181.5 ± 27.1	0.7 ± 0.8
HA 4	183.3 ± 20.5	-2.4 ± 0.6	192.8 ± 41.5	-1.7 ± 2.9

ms = milliseconds; μV = microvolts

The P2 amplitude measurements for almost half of the normal hearing children were also found to be statistically significant [NH 2: ($p<0.05$), $t(3) = 4.33$, $p = 0.023$; TiS 1: ($p<0.05$), $t(3) = 6.36$, $p = 0.008$; TiS 3: ($p<0.05$), $t(4) = 5.13$, $p = 0.007$; TiS 4: ($p<0.05$), $t(3) = 24.5$, $p = <0.0001$]. The remaining four children matched for chronological age [NH 1: ($p<0.05$), $t(3) = 2.18$, $p = 0.118$; NH 3: ($p<0.05$), $t(3) = 0.647$, $p = 0.564$; NH 4: ($p<0.05$), $t(3) = 1.98$, $p = 0.142$; NH 5: ($p<0.05$), $t(3) = 1.11$, $p = 0.347$] and two children matched for "time-in-sound" [TiS 2: ($p<0.05$), $t(3) = 0.311$, $p = 0.776$; TiS 5: ($p<0.05$), $t(3) = -1.15$, $p = 0.332$] had no significant differences recorded in the P2 amplitudes when evoked by different stimuli.

3.1.4 N2 Latency and Amplitude

The N2 latency and amplitude mean and standard deviation measurements recorded in response to the 193 Hz pure-tone and the /ba/ recorded consonant-vowel speech stimulus are displayed in Table 6 for children with cochlear implants, normal hearing, and the child with hearing aids.

Table 6: N2 Peak Latency and Amplitude Measurements

The N2 latency and amplitude measurements recorded in response to a 193 Hz pure-tone and a recorded /ba/ speech stimulus, presented at 70 dB SPL, for children with cochlear implants (CI), chronologically age matched normal hearing children (NH), time-in-sound age matched normal hearing children (TiS), and a chronologically age matched child with hearing aids (HA). Means and standard deviations are shown (n = 4 replicates of 256 averages).

Participant	193 Hz – N2 Latency (ms)	193 Hz – N2 Amplitude (μV)	/ba/ - N2 Latency (ms)	/ba/ - N2 Amplitude (μV)
CI 1	302.5 ± 4.0	-2.1 ± 0.4	267 ± 8.8	-6.3 ± 1.0
CI 2	215.3 ± 6.2	-1.1 ± 3.3	281.5 ± 41.3	-5.8 ± 3.3
CI 3	267.8 ± 15.2	-3.2 ± 0.5	275 ± 15.2	-3.9 ± 0.9
CI 4	227.8 ± 12.9	-3.6 ± 1.6	235.8 ± 9.0	-9.2 ± 2.3
CI 5	307.8 ± 3.7	-1.0 ± 0.9	256.8 ± 15.5	-1.1 ± 0.4
NH 1	328.5 ± 25.8	-2.1 ± 0.7	233.3 ± 19.1	-3.4 ± 0.7
NH 2	309.8 ± 44.9	-1.4 ± 0.4	222 ± 1.6	-3.9 ± 0.3
NH 3	258.2 ± 53.7	-2.2 ± 1.7	223.5 ± 12.4	-4.4 ± 1.2
NH 4	250.8 ± 16.2	-1.1 ± 0.9	225.3 ± 8.5	-5.3 ± 1.3
NH 5	229.8 ± 7.6	-1.9 ± 0.4	237.5 ± 8.6	-3.6 ± 0.8
TiS 1	379 ± 16.4	-3.1 ± 0.2	287.3 ± 77.6	-2.9 ± 0.6
TiS 2	279.3 ± 33.8	-0.4 ± 1.0	256.5 ± 22.9	-4.4 ± 0.7
TiS 3	281.2 ± 22.1	-1.6 ± 0.7	264.4 ± 47.3	-4.6 ± 1.9
TiS 4	307.3 ± 45.4	-0.7 ± 0.7	221.3 ± 15.4	-3.8 ± 0.5
TiS 5	265.3 ± 9.1	-1.6 ± 0.4	244.5 ± 39.2	-2.8 ± 0.6
HA 4	208 ± 28.2	-4.7 ± 1.1	235.5 ± 16.8	-5.6 ± 0.2

ms = milliseconds; μV = microvolts

Two children with cochlear implants had statistically significant differences [CI 1: ($p<0.05$), $t(3) = 6.19$, $p = 0.009$; CI 5: ($p<0.05$), $t(3) = 5.49$, $p = 0.012$] in their N2 latency measurements when CAEPs were evoked using a pure-tone and a speech stimulus. The other three children with cochlear implants [CI 2: ($p<0.05$), $t(3) = -3.14$, $p = 0.052$; CI 3: ($p<0.05$), $t(3) = -0.52$, $p = 0.641$; CI 4: ($p<0.05$), $t(3) = -1.08$, $p = 0.361$] and the child with hearing aids [HA 4: ($p<0.05$), $t(3) = -1.33$, $p = 0.275$] resulted in no significant differences between the two N2 latency measurements.

Two chronologically age-matched children with normal hearing were also found to have statistically significant differences [NH 1: ($p<0.05$), $t(3) = 4.28$, $p = 0.023$; NH 2: ($p<0.05$), $t(3) = 3.76$, $p = 0.033$] in their N2 latencies, while the other three children [NH 3: ($p<0.05$), $t(3) = 0.71$, $p = 0.527$; NH 4: ($p<0.05$), $t(3) = 2.72$, $p = 0.072$; NH 5: ($p<0.05$), $t(3) = -2.82$, $p = 0.067$] had no significant differences. There were no statistically significant differences found between the two N2 latency measurements recorded for any of the normal hearing children matched for “time-in-sound” [TiS 1: ($p<0.05$), $t(3) = 2.53$, $p = 0.086$; TiS 2: ($p<0.05$), $t(3) = 2.33$, $p = 0.102$; TiS 3: ($p<0.05$), $t(4) = 0.94$, $p = 0.402$; TiS 4: ($p<0.05$), $t(4) = 2.93$, $p = 0.061$; TiS 5: ($p<0.05$), $t(3) = 1.09$, $p = 0.356$].

The N2 amplitudes for four of the children with hearing instruments were found to have no statistically significant differences [CI 2: ($p<0.05$), $t(3) = 1.68$, $p = 0.191$; CI 3: ($p<0.05$), $t(3) = 1.92$, $p = 0.15$; CI 5: ($p<0.05$), $t(3) = 0.271$, $p = 0.804$; HA 4: ($p<0.05$), $t(3) = 1.50$, $p = 0.23$]. Two children with cochlear implants [CI 1: ($p<0.05$), $t(3) = 10.5$, $p = 0.002$; CI 4: ($p<0.05$), $t(3) = 4.79$, $p = 0.017$] recorded N2 amplitudes that were significantly different when elicited by the pure-tone and speech stimuli.

The N2 amplitude measurements elicited by the two stimuli were found to be significantly different for the majority of the normal hearing children matched for chronological age [NH 2: ($p<0.05$), $t(3) = 8.06$, $p = 0.004$; NH 4: ($p<0.05$), $t(3) = 8.17$, $p = 0.004$; NH 5: ($p<0.05$), $t(3) = 3.39$, $p = 0.043$] and matched for "time-in-sound" [TiS 2: ($p<0.05$), $t(3) = 5.89$, $p = 0.01$; TiS 3: ($p<0.05$), $t(4) = 4.16$, $p = 0.014$; TiS 4: ($p<0.05$), $t(3) = 5.71$, $p = 0.011$; TiS 5: ($p<0.05$), $t(3) = 3.71$, $p = 0.034$]. Only three children [NH 1: ($p<0.05$), $t(3) = 2.04$, $p = 0.135$; NH 3: ($p<0.05$), $t(3) = 1.75$, $p = 0.179$; TiS 1: ($p<0.05$), $t(3) = -0.77$, $p = 0.497$] were shown to have no statistically significant differences between the two N2 amplitude measurements.

3.2 Waveform Morphology – 193 Hz

The peak latencies and amplitudes of the CAEP waveforms of the children with cochlear implants were compared to the corresponding two groups of children with normal hearing matched for chronological age and matched for "time-in-sound", as well as the child with hearing aids, where applicable. These comparative peaks for each group of children (for example, CI 4, NH 4, TiS 4, and HA 4) are displayed in order in Figures 15, 16, 17, 18, and 19. One-way repeated measures ANOVAs were completed to analyse the four peaks recorded in the waveforms in response to the 193 Hz pure-tone stimulus.

3.2.1 CI 1

ANOVAs were completed and found statistically significant differences for all the peaks (P1: [$F(2, 9) = 93.45$, $p < 0.0001$]; N1: [$F(2, 9) = 6.42$, $p = 0.02$]; P2: [$F(2, 9) = 16.24$, $p = 0.001$]; N2: [$F(2, 9) = 19.1$, $p = 0.0006$]) between CI 1, NH 1, and TiS 1 in the peak latencies of the waveforms elicited by the 193 Hz pure-tone stimulus, as displayed in Figure 15.

Post-hoc pair-wise multiple comparisons procedure using the Tukey HSD ("honestly significant difference") test revealed that the P1 and P2 latency measurements of the child with cochlear implants were significantly different from both the chronologically age matched normal hearing child and the time-in-sound matched normal hearing child. The Tukey HSD test showed no statistically

significant difference between the P1 and P2 latency measurements for the two normal hearing children.

Further analysis through the use of Tukey tests revealed significant differences between the child with a cochlear implant and the child matched for “time-in-sound” for the N1, and N2 latencies. Additional significant differences were found between the two normal hearing children for the N2 latency measurements.

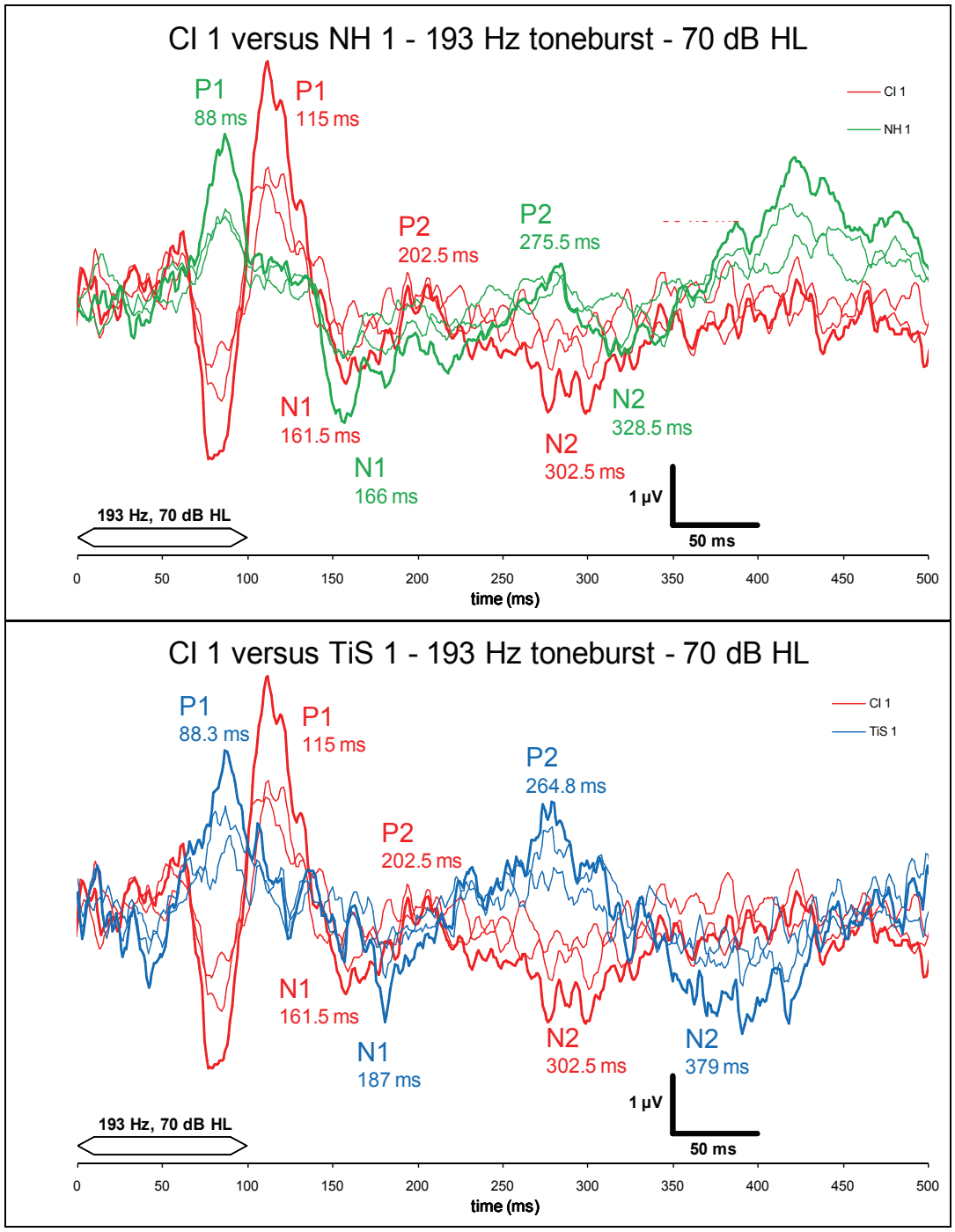


Figure 15: The CAEP waveforms, recorded in response to 193 Hz pure-tone stimulus, of CI 1 overlaid by the waveform recorded from NH 1 (top) and from TiS 1 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

ANOVA tests revealed no statistically significant differences between the three children's P1 [$F(2, 9) = 0.97$, $p = 0.42$] and N1 [$F(2, 9) = 1.93$, $p = 0.2$] amplitude measurements elicited in response to the 193 Hz pure-tone. Statistical differences were found between the P2, [$F(2, 9) = 5.15$, $p = 0.03$], and N2, [$F(2, 9) = 5.21$, $p = 0.03$], amplitudes recorded. However, further analysis using Tukey HSD tests found no significant difference between the P2 amplitudes of any of the three children. In addition, the statistical difference found in the N2 amplitude measurements was between the two normal hearing children, with no significant difference between CI 1 with either NH 1 or TiS 1.

3.2.2 CI 2

The peak latencies, as shown in Figure 16, measured in response to the pure-tone stimulus for CI 2, NH 2, and TiS 2 were found to be not statistically significant, [$F(2, 9) = 1.88$, $p = 0.21$], for P1 when analysed. However, ANOVA results found statistically significant differences in the peak latency measurements for N1, [$F(2, 9) = 6.35$, $p = 0.02$], P2, [$F(2, 9) = 9.08$, $p = 0.006$] and N2, [$F(2, 9) = 8.1$, $p = 0.01$], between these three children.

Tukey HSD tests were used to further analyse the latency results and for N1 found that there was a significant difference between the child with a cochlear implant and the normal hearing child matched for "time-in-sound". In contrast, analysis of the P2 and N2 latency measurements both showed a significant difference

between the child with a cochlear implant and the chronologically age-matched child with normal hearing.

ANOVA tests revealed statistically significant differences in the P1, [$F(2, 9) = 14.48$, $p = 0.002$], N1, [$F(2, 9) = 7.98$, $p = 0.01$], P2, [$F(2, 9) = 12.16$, $p = 0.003$], amplitude measurements between children CI 2, NH 2, and TiS 2, when elicited by a 193 Hz pure-tone. The N2 peak [$F(2, 9) = 0.35$, $p = 0.71$] was found to have no significant differences.

No significance was found between the P1, N1, or P2 amplitudes for either of the children with normal hearing when further analysis was completed using the Tukey HSD test. Significant differences were found between the P1 amplitudes of the child with cochlear implants and both normal hearing children. The N1 amplitude measurements were only significantly different between the child with cochlear implants and the normal hearing child matched for chronological age. In contrast, the P2 amplitudes were found to have statistically significant differences between the child with cochlear implants and the normal hearing child matched for “time-in-sound”.

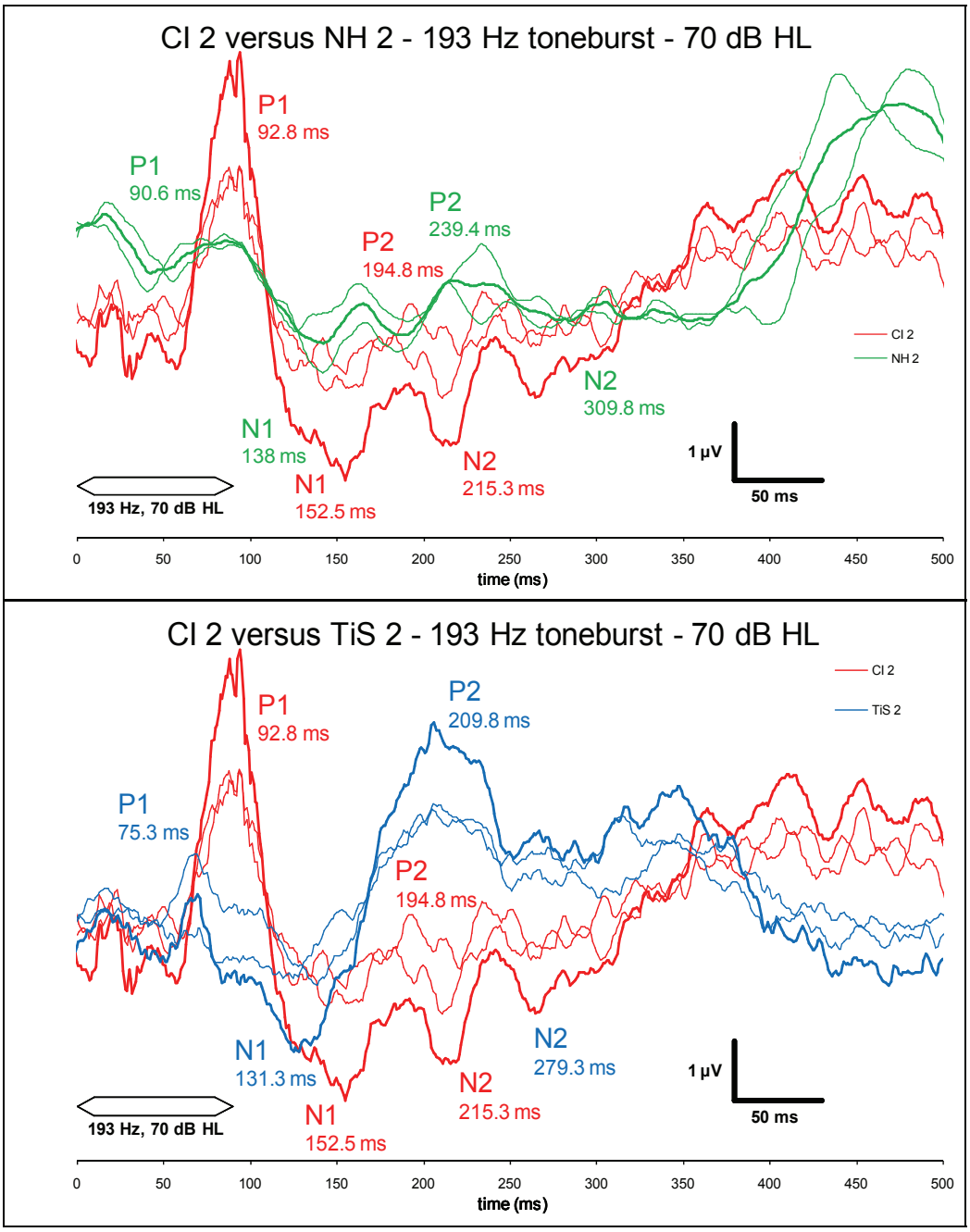


Figure 16: The CAEP waveforms, recorded in response to 193 Hz pure-tone stimulus, of CI 2 overlaid by the waveform recorded from NH 2 (top) and from TiS 2 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms. The waveforms for NH2 have been smoothed with a 20 ms running-point average to remove intrusive 50 Hz interference.

3.2.3 CI 3

One-way repeated measures ANOVAs were completed to analyse the waveforms of CI 3, NH 3, and TiS 3, as shown in Figure 17. No statistically significant differences were found for the peak latencies of N1 [$F(2, 9) = 2.7$, $p = 0.12$]. The other three peaks, P1, [$F(2, 9) = 5.45$, $p = 0.03$], P2, [$F(2, 9) = 79.57$, $p < 0.0001$], and N2, [$F(2, 9) = 9.51$, $p = 0.006$], were found to have significant differences in latency measurements between the three children when evoked by the pure-tone stimulus.

Tukey HSD tests used to analyse the P1 latencies revealed a statistically significant difference between the child with cochlear implants and the normal hearing child matched for "time-in-sound". Significant differences were also found between both the normal hearing children. P2 latency measurements were found to be statistically different between all three children, while N2 latencies were found to only have significant differences between the two children with normal hearing.

The amplitudes were also analysed using ANOVAs and found significant differences between these measurements for children CI 3, NH 3, and TiS 3 for the peaks P1, [$F(2, 9) = 40.9$, $p < 0.0001$], and N1, [$F(2, 9) = 14.38$, $p = 0.002$].

No significant differences were found for the last two peaks, P2, [$F(2, 9) = 1.47$, $p = 0.28$], and N2, [$F(2, 9) = 2.6$, $p = 0.13$].

Statistically significant differences were found between all three children for the P1 amplitudes, when further analysed using the Tukey HSD test. N1 amplitude measurements were also found to significantly differ between the child with a cochlear implant and both children with normal hearing, with no difference found between the two children with normal hearing.

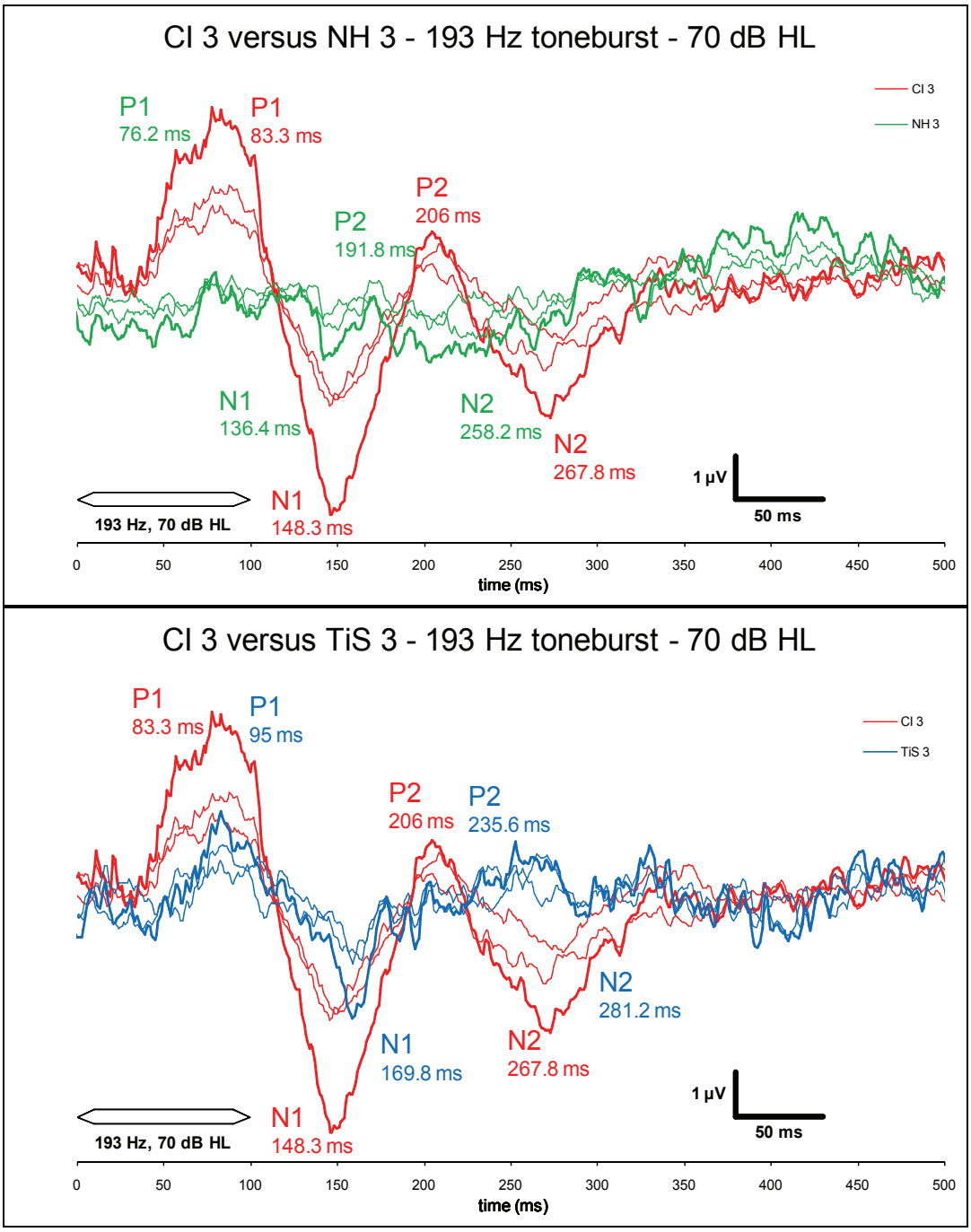


Figure 17: The CAEP waveforms, recorded in response to 193 Hz pure-tone stimulus, of CI 3 overlaid by the waveform recorded from NH 3 (top) and from TiS 3 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

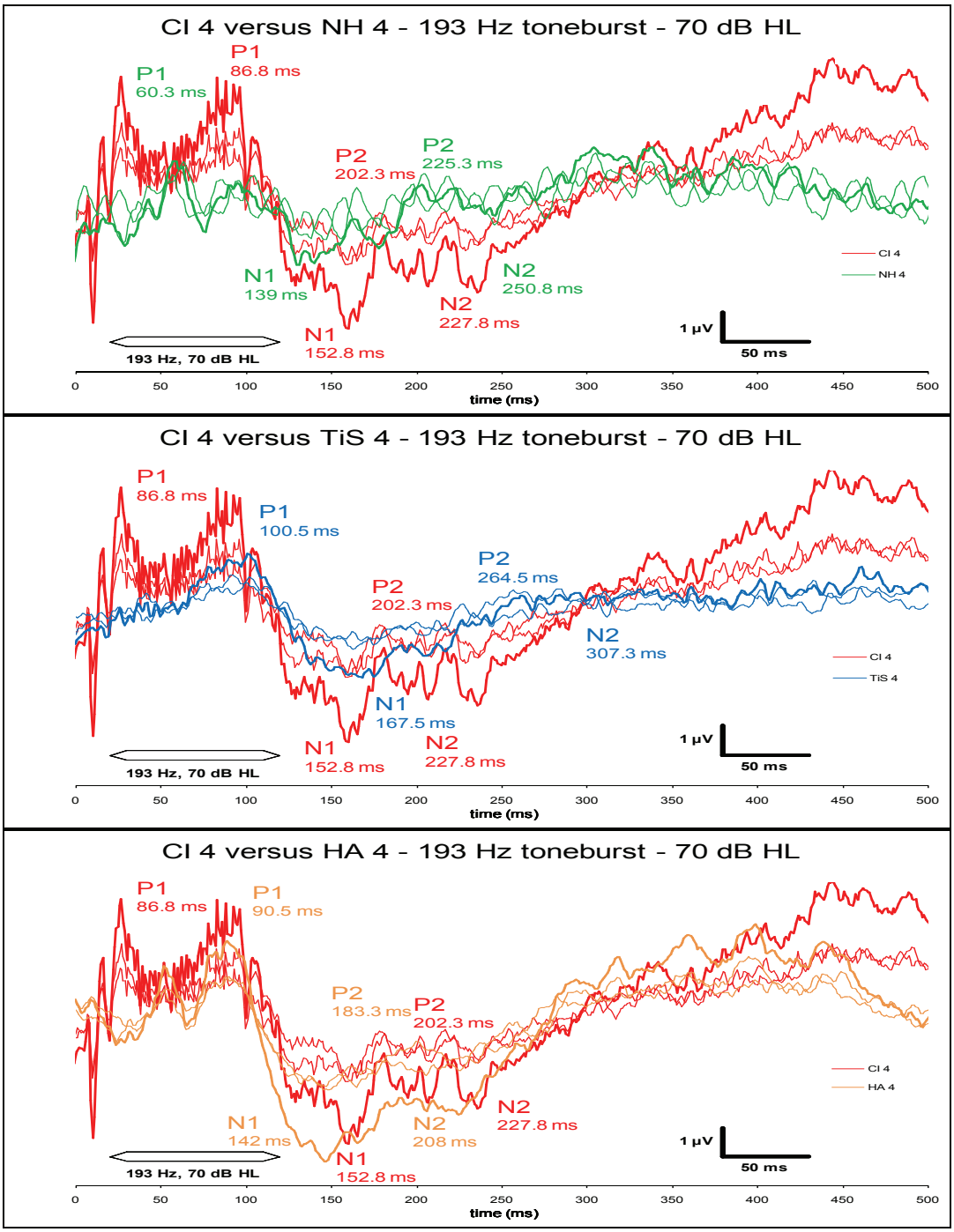


Figure 18: The CAEP waveforms, recorded in response to 193 Hz pure-tone stimulus, of CI 4 overlaid by the waveform recorded from NH 4 (top), TiS 4 (middle), and HA 4 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

3.2.4 CI 4

ANOVAs revealed there were statistically significant differences between the latency measurements recorded in response to the 193 Hz pure-tone stimulus between CI 4, NH 4, TiS 4, and HA 4, as displayed in Figure 18. All four peaks, P1, [$F(3, 12) = 31.93$, $p < 0.0001$], N1, [$F(3, 12) = 6.76$, $p = 0.006$], and P2, [$F(3, 12) = 13.48$, $p = 0.0004$], N2, [$F(3, 12) = 8.97$, $p = 0.002$], were found to be significantly different in latency measurements.

Tukey HSD tests were used to further analyse the results. P1 latencies were found to differ significantly between the child with a cochlear implant and both children with normal hearing. The chronologically age-matched child with normal hearing was also found to have a significantly different P1 latency from the other child with normal hearing as well as the child with hearing aids. Significant differences in P2 latencies were found between the child with a cochlear implant and the child matched for “time-in-sound”, as well as between the child with hearing aids and both normal hearing children.

The latency measurements of the negative peaks were only found to significantly differ between the normal hearing child matched for “time-in-sound” with the other normal hearing child and the child with hearing aids for N1, and with the two children with hearing instruments for N2.

No significant differences were found between either of the children with hearing instruments for any of the five peak latency measurements.

Significant differences in amplitude measurements were also found for all four peaks, P1, [$F(3, 12) = 15.35$, $p = 0.0002$], N1, [$F(3, 12) = 13.74$, $p = 0.0003$], P2, [$F(3, 12) = 14.69$, $p = 0.0003$], and N2, [$F(3, 12) = 11.95$, $p = 0.0006$] between the four children when analysed using ANOVAs. The amplitude measurements of P1 were found to differ significantly between the child with a cochlear implant and all three other children. P2 amplitude measurements were found to significantly differ between the child with a cochlear implant and the chronologically age-matched child with normal hearing as well as between the child with hearing aids and both normal hearing children.

The two negative peaks were further analysed using Tukey tests and were found to have statistically significant differences between the children with hearing instruments and the children with normal hearing. No differences were revealed between the two children with hearing instruments or between the two children with normal hearing.

3.2.5 CI 5

The CAEP waveforms, elicited by the 193 Hz pure-tone stimulus, of CI 5, NH 5, and TiS 5 are displayed in Figure 19. The peak latencies were measured and analysed and no significant differences were found for P1, [$F(2, 9) = 2.74$, $p = 0.12$], and N1, [$F(2, 9) = 2.09$, $p = 0.18$], between the three children. ANOVA results showed that there were statistically significant differences in the latency measurements for the last two peaks, P2, [$F(2, 9) = 12.76$, $p = 0.002$] and N2, [$F(2, 9) = 118.26$, $p < 0.0001$].

Tukey HSD tests revealed statistically significant differences between the child with a cochlear implant and both normal hearing children for all of the last three peaks. A significant difference was also found between the two normal hearing children for the N2 peak latency measurements.

ANOVAs revealed no significant differences in amplitude measurements for half of the peaks, including N1, [$F(2, 9) = 0.69$, $p = 0.53$], P2, [$F(2, 9) = 1.3$, $p = 0.32$] and N2, [$F(2, 9) = 2.13$, $p = 0.017$]. P1 amplitudes were the only measurements to show a statistical difference [$F(2, 9) = 4.75$, $p = 0.04$] between the three children. However, on further analysis, using the Tukey HSD test, no statistically significant differences were found between any of the three children.

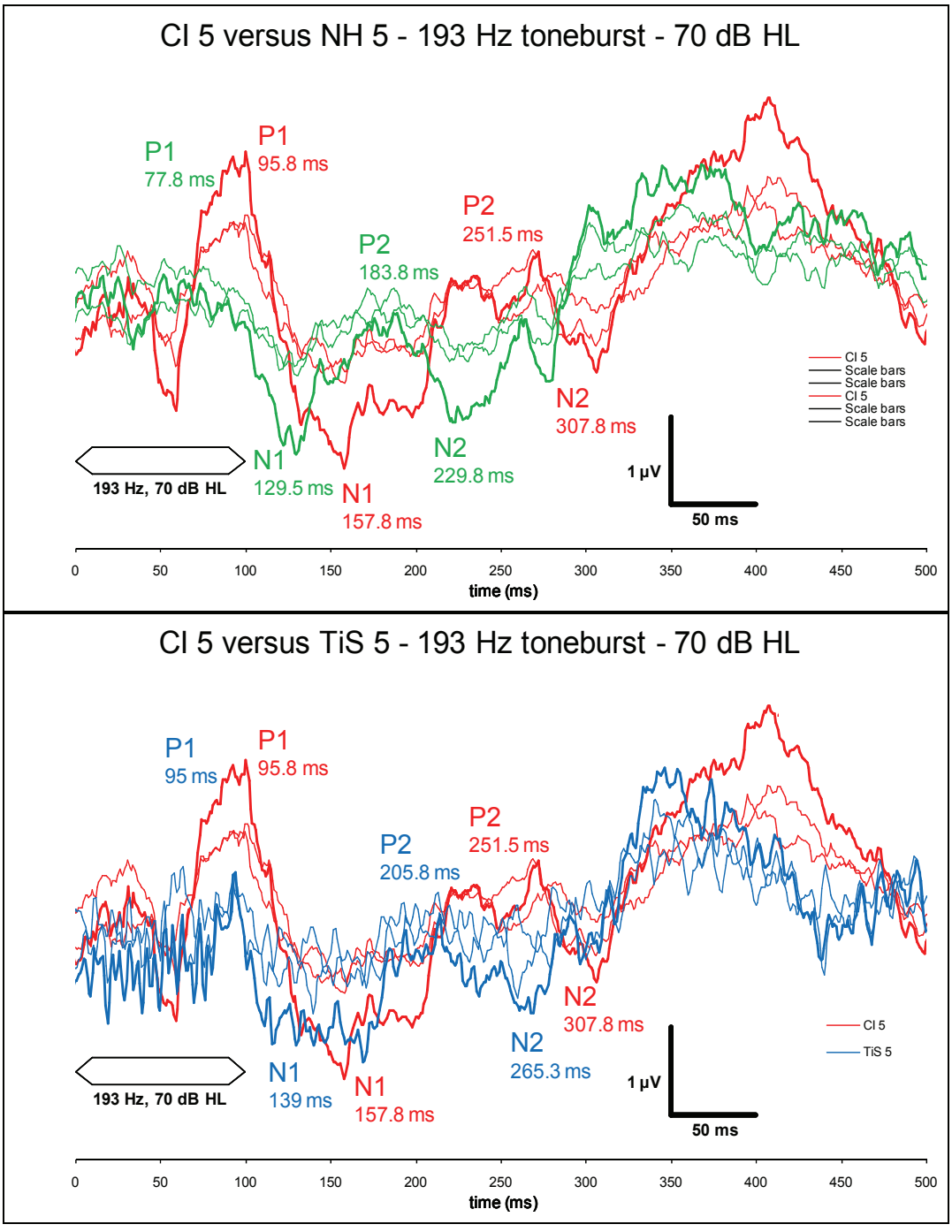


Figure 19: The CAEP waveforms, recorded in response to 193 Hz pure-tone stimulus, of CI 5 overlaid by the waveform recorded from NH 5 (top) and from TiS 5 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

3.3 Waveform Morphology – /ba/

The CAEP waveforms recorded in response to the speech stimulus /ba/ were measured and evaluated between the children with cochlear implants and the corresponding chronologically age-matched children with normal hearing and the children with normal hearing matched for "time-in-sound", as well as the child with hearing aids, where applicable. These comparative waveforms for each group of children (for example, CI 4, NH 4, TiS 4, and HA 4) are displayed in order in Figures 20, 21, 22, 23, and 24. One-way repeated measures ANOVAs were completed to analyse all five peak's latency and amplitude measurements recorded in response to the /ba/ speech stimulus.

3.3.1 CI 1

The peak latencies elicited by the speech stimulus of CI 1, NH 1, and TiS 1, as shown in Figure 20, were compared using a one-way repeated measures ANOVA. No statistically significant differences were found for any of the peak latency measurements, including P1, [$F(2, 9) = 1.56$, $p = 0.26$], N1, [$F(2, 9) = 1.46$, $p = 0.28$], P2, [$F(2, 9) = 3.6$, $p = 0.07$], and N2, [$F(2, 9) = 1.38$, $p = 0.3$]. No statistical differences were found between the three children's peak amplitude measurements for peaks P1, [$F(2, 9) = 30.19$, $p = 0.0001$], or N1, [$F(2, 9) = 1.79$, $p = 0.22$]. The other two peaks, P2, [$F(2, 9) = 5.36$, $p = 0.03$], and N2, [$F(2, 9) = 22.19$, $p = 0.0003$], were found to have statistically significant differences.

Further analysis through the use of Tukey HSD tests revealed that the P2 amplitude measurements were significantly different between the child with a cochlear implant and the normal hearing child matched for chronological age. Statistical differences were also found between the child with a cochlear implant and both normal hearing children for N2 amplitude measurements.

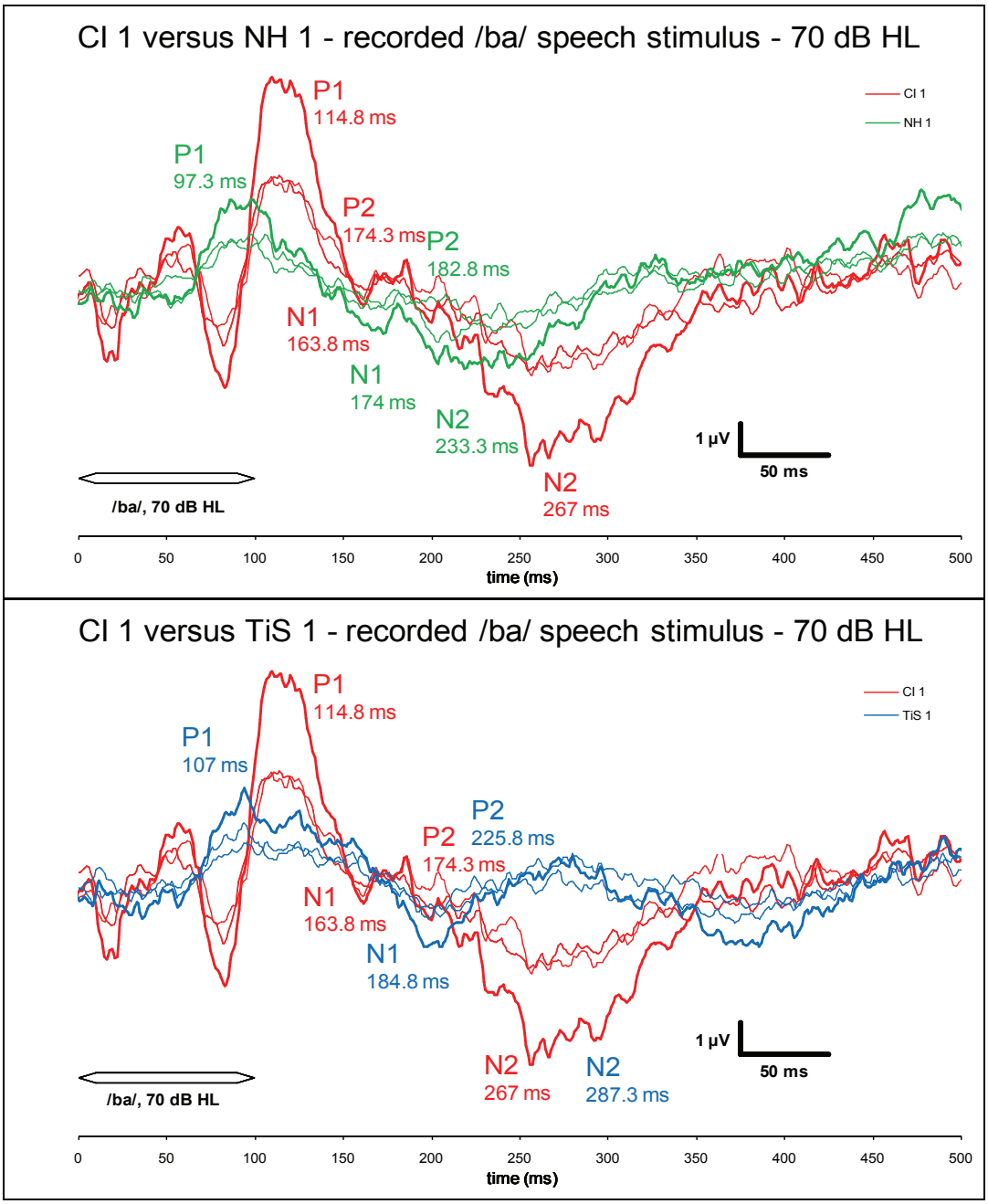


Figure 20: The CAEP waveforms, recorded in response to the recorded /ba/ speech stimulus, of CI 1 overlaid by the waveform recorded from NH 1 (top) and from TiS 1 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

3.3.2 CI 2

ANOVA tests were completed to analyse the waveforms, as displayed in Figure 21, of CI 2, NH 2, and TiS 2. No statistically significant differences in latency measurements were found for the first three peaks, P1, [$F(2, 9) = 3.41$, $p = 0.08$], N1, [$F(2, 9) = 3.74$, $p = 0.07$], and P2, [$F(2, 9) = 0.48$, $p = 0.63$]. The final peak, N2, [$F(2, 9) = 4.81$, $p = 0.04$], was found to have statistically significant differences.

Tukey HSD tests revealed that the N2 latency measurements were significantly different between the child with cochlear implants and the chronologically age-matched child with normal hearing.

ANOVA tests revealed that P2, [$F(2, 9) = 8.86$, $p = 0.007$], was the only peak amplitude measurement that was statistically significant. A Tukey test found that the P2 amplitude for the normal hearing child matched for "time-in-sound" was significantly different to both the child with a cochlear implant and the other normal hearing child. The other three peaks, P1, [$F(2, 9) = 0.45$, $p = 0.65$], N1, [$F(2, 9) = 0.25$, $p = 0.78$], and N2, [$F(2, 9) = 1.06$, $p = 0.39$], were not found to be statistically different.

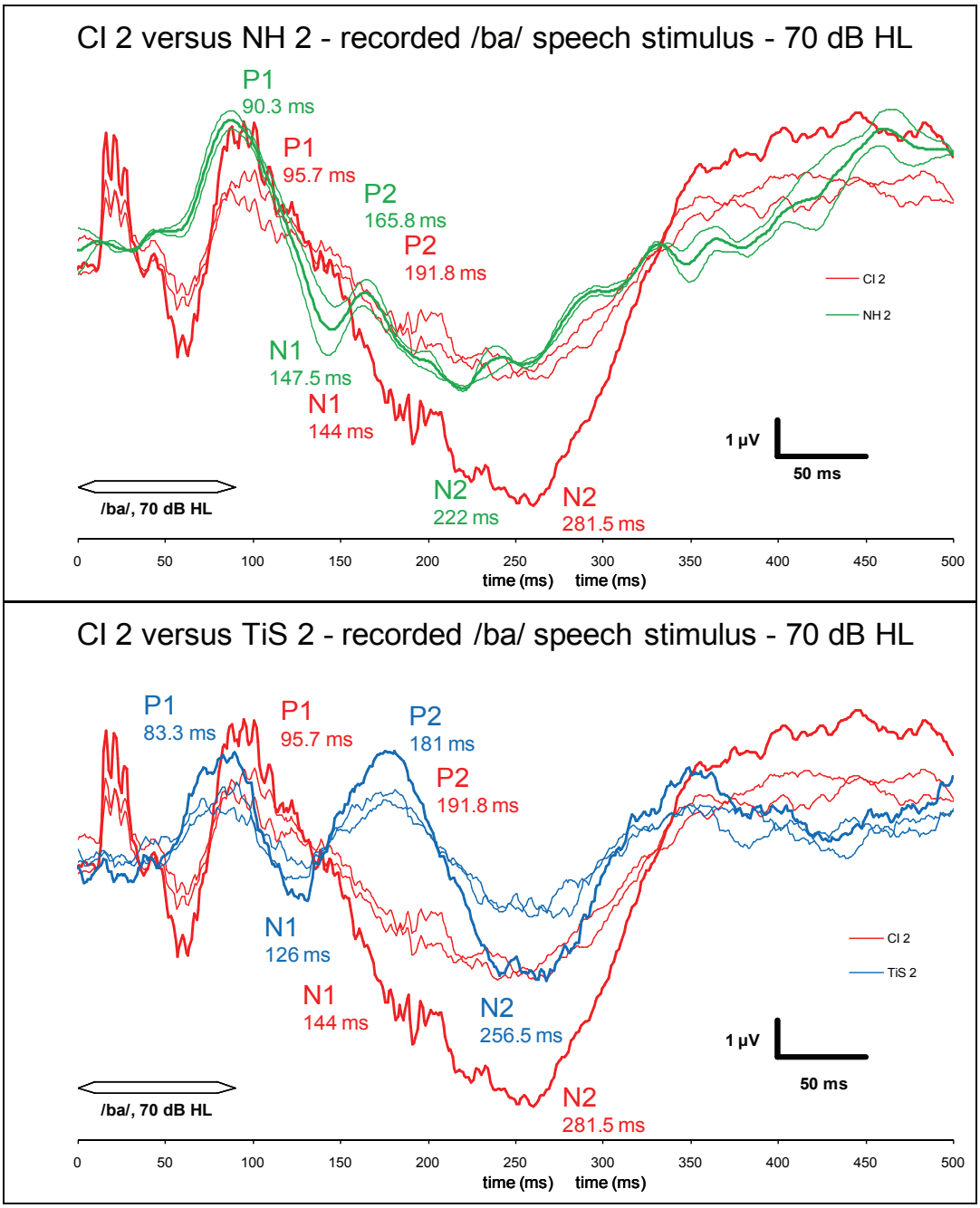


Figure 21: The CAEP waveforms, recorded in response to the recorded /ba/ speech stimulus, of CI 2 overlaid by the waveform recorded from NH 2 (top) and from TiS 2 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms. The waveforms for NH2 have been smoothed with a 20 ms running-point average to remove intrusive 50 Hz interference.

3.3.3 CI 3

A one-way repeated measures ANOVA revealed there was no statistically significant difference of the peak latency measurements recorded in response to the speech stimulus for N1, [$F(2, 9) = 2.58$, $p = 0.13$], and P2, [$F(2, 9) = 1.02$, $p = 0.4$]. The peak latency measurements of the three children, CI 3, NH 3, and TiS 3 were found to have a statistically significant difference for P1, [$F(2, 9) = 7.86$, $p = 0.01$], and N2, [$F(2, 9) = 6.17$, $p = 0.02$]. Further analysis revealed that the child with a cochlear implant recorded significantly different latencies from the chronologically age-matched child with normal hearing for both peaks P1 and N2. These waveforms are all displayed in Figure 22.

The peak amplitudes of P1, [$F(2, 9) = 0.4$, $p = 0.68$], N1, [$F(2, 9) = 0.59$, $p = 0.57$], P2, [$F(2, 9) = 3.61$, $p = 0.07$], and N2, [$F(2, 9) = 0.99$, $p = 0.41$], were found to have no significant difference between the three children when recorded in response to the /ba/ speech stimulus.

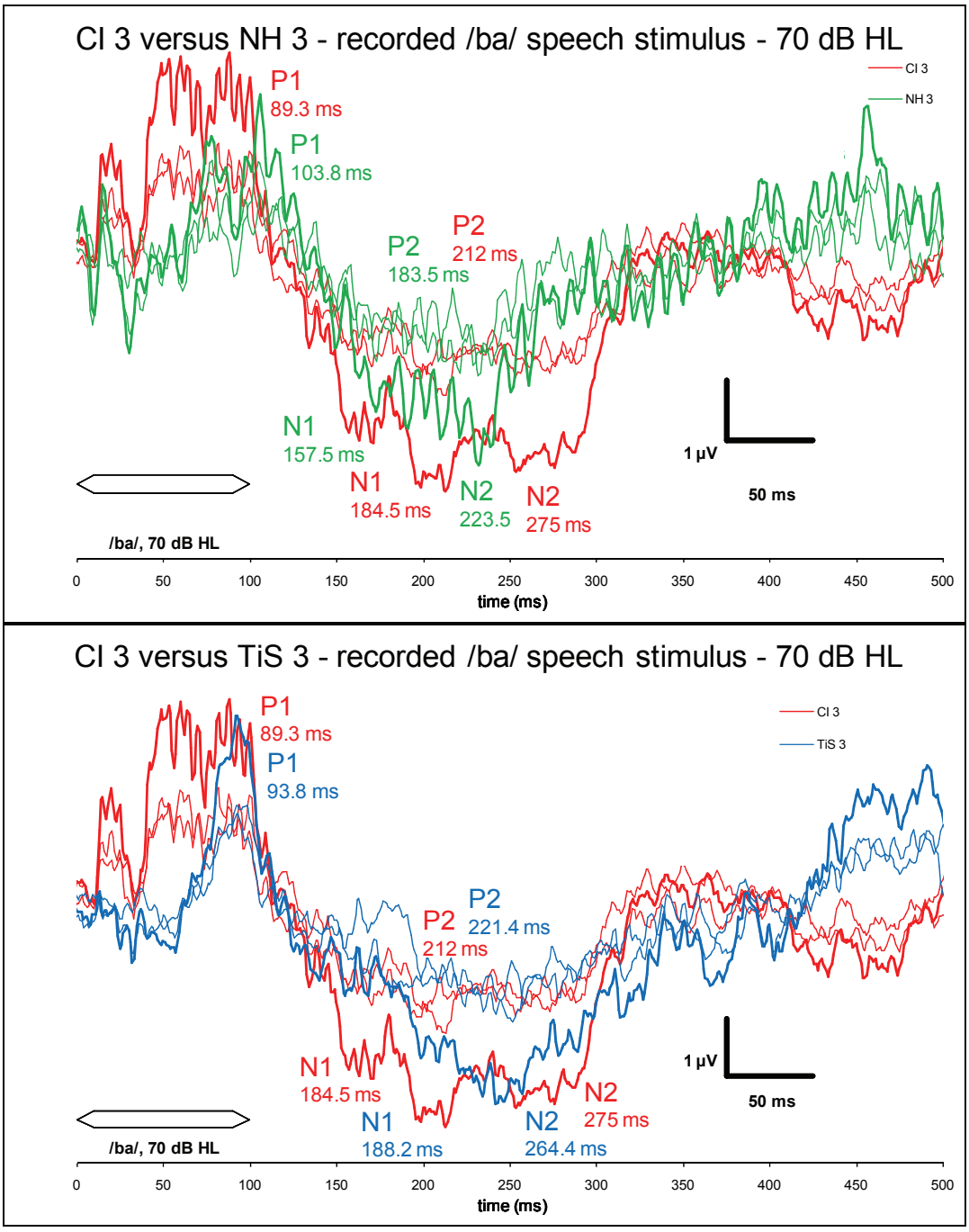


Figure 22: The CAEP waveforms, recorded in response to the recorded /ba/ speech stimulus, of CI 3 overlaid by the waveform recorded from NH 3 (top) and from TiS 3 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

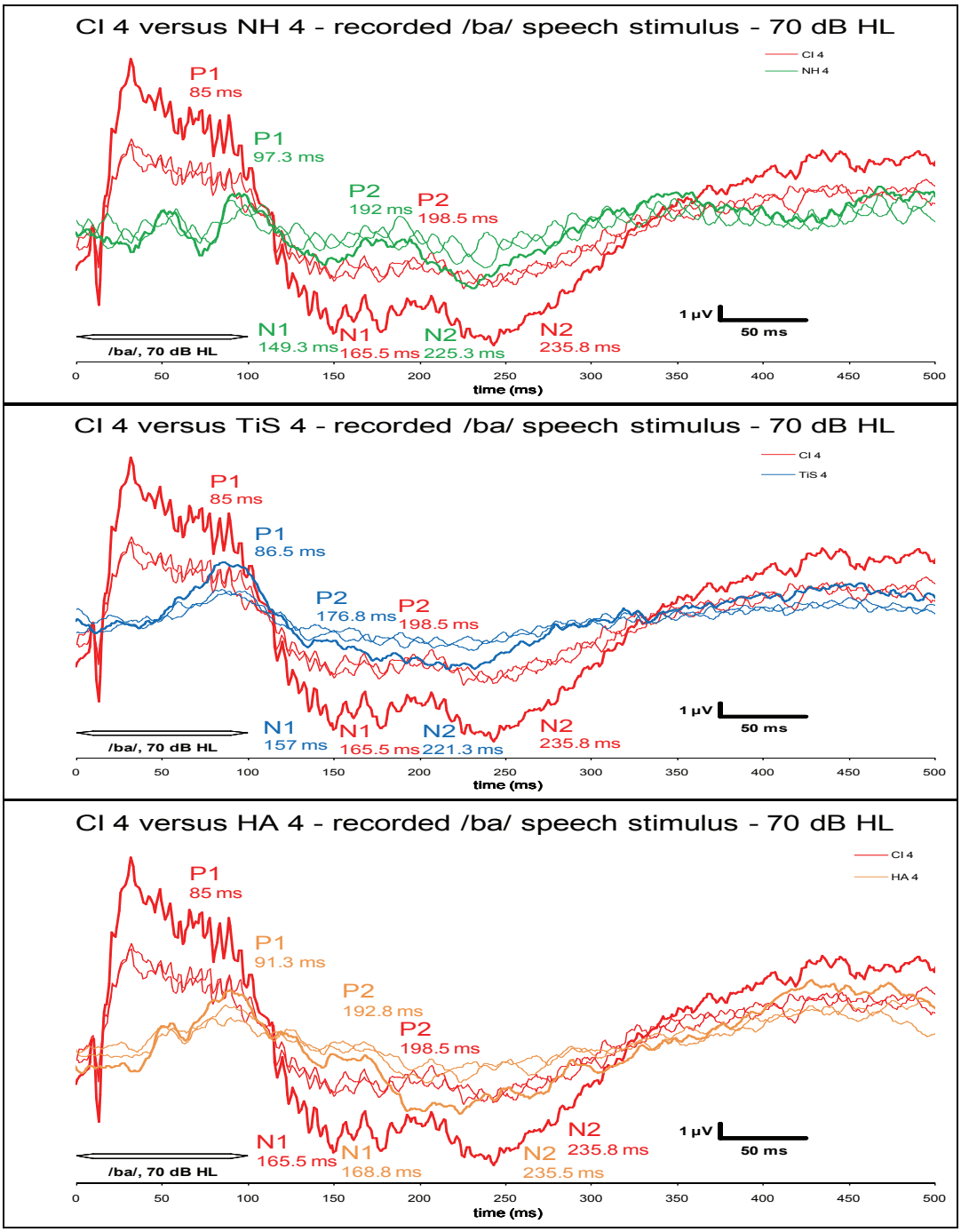


Figure 23: The CAEP waveforms, recorded in response to the recorded /ba/ speech stimulus, of CI 4 overlaid by the waveform recorded from NH 4 (top), TiS 4 (middle), and HA 4 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

3.3.4 CI 4

The waveforms elicited by the consonant-vowel speech stimulus of CI 4, NH 4, TiS 4, and HA 4, shown in Figure 23, were analysed using ANOVA tests. No statistically significant differences were found for the first four peak latencies P1, [$F(3, 12) = 3.26$, $p = 0.06$], N1, [$F(3, 12) = 0.72$, $p = 0.56$], P2, [$F(3, 12) = 0.46$, $p = 0.72$], or N2, [$F(3, 12) = 1.28$, $p = 0.33$], between the four children.

An ANOVA found that the peak amplitude measurements of all four peaks, P1, [$F(3, 12) = 13.4$, $p = 0.0004$], N1, [$F(3, 12) = 9.92$, $p = 0.001$], P2, [$F(3, 12) = 6.32$, $p = 0.008$], and N2, [$F(3, 12) = 11.76$, $p = 0.0007$], were statistically significant for the four children. Tukey tests revealed that the child with a cochlear implant had significantly different P1, N1, and N2 amplitude measurements to the three other children. The child with a cochlear implant also had statistically significant differences from the two normal hearing children in the P2 amplitude measurements.

3.3.5 CI 5

The peak latencies elicited in response to the speech stimulus of CI 5, NH 5, and TiS 5 were found to be not significantly different for any of the peaks (P1, [$F(2, 9) = 0.02$, $p = 0.98$], N1, [$F(2, 9) = 0.14$, $p = 0.87$], P2, [$F(2, 9) = 0.92$, $p = 0.43$], N2, [$F(2, 9) = 0.62$, $p = 0.56$]). Furthermore, the amplitude measurements for P1 [$F(2, 9) = 0.92$, $p = 0.43$] were found to have no statistically significant difference between the three children, whose waveforms are all displayed in Figure 24. A statistical difference in amplitude was found for peaks N1, [$F(2, 9) = 5.2$, $p = 0.03$], P2, [$F(2, 9) = 20.78$, $p = 0.0004$], and N2, [$F(2, 9) = 18.17$, $p = 0.0007$], between the three children. Tukey tests revealed that the child with a cochlear implant had significantly different N1 amplitudes from the normal hearing child matched for chronological age as well as P2 and N2 amplitudes from both children with normal hearing.

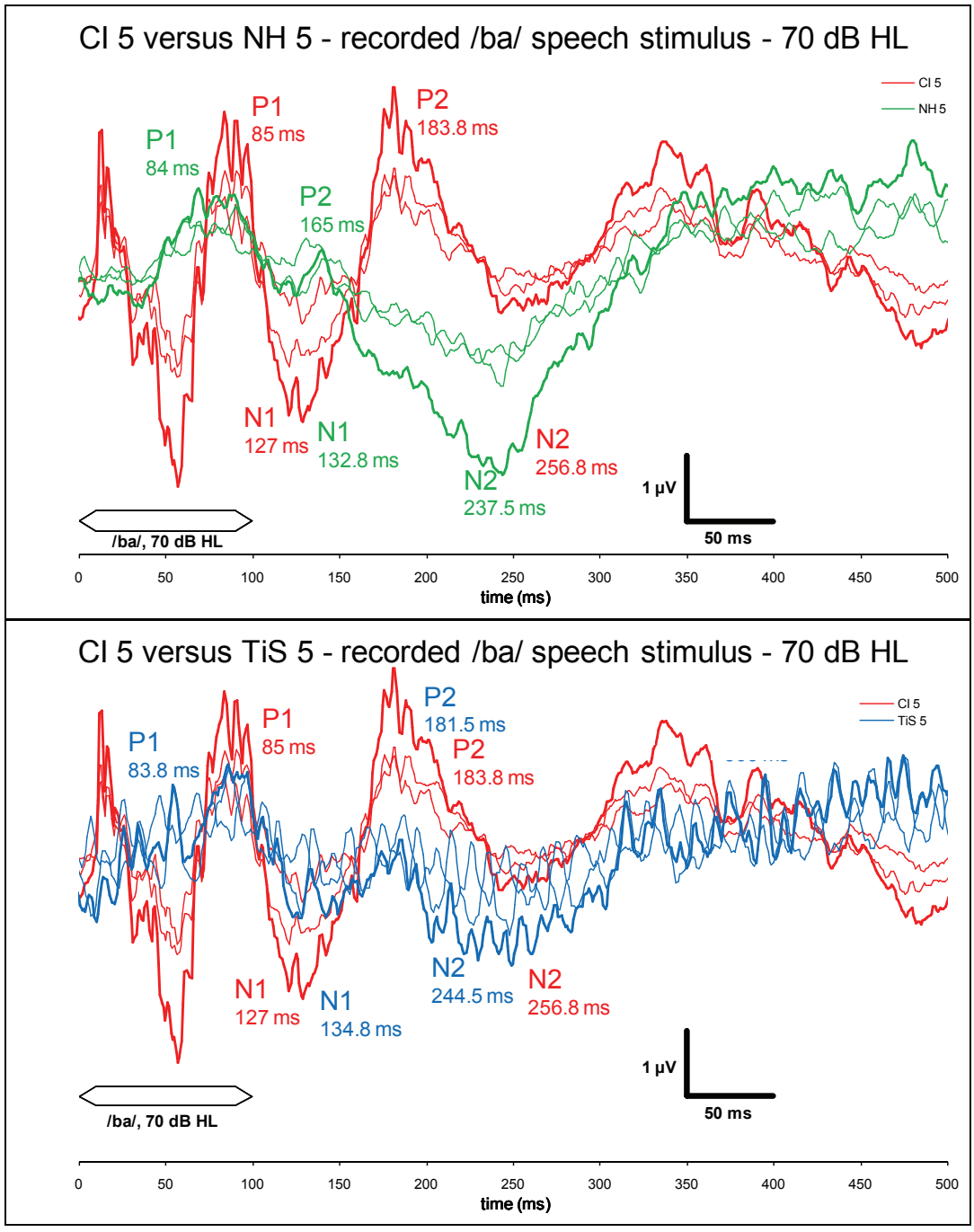


Figure 24: The CAEP waveforms, recorded in response to the recorded /ba/ speech stimulus, of CI 5 overlaid by the waveform recorded from NH 5 (top) and from TiS 5 (bottom). The bold line is the totalled waveforms. All four peaks and their mean latencies are marked for each child's waveforms.

Chapter 4

Discussion

4.1 Summary of Main Findings

The present study examined and compared the CAEP waveforms of five children who received cochlear implants prior to 3.5 years of age to five chronologically age-matched children with normal hearing, and five children with normal hearing who were matched for "time-in-sound", as well as an individual child who received hearing aids prior to 3.5 years of age. The latencies and amplitudes of the three positive and two negative peaks of the CAEP waveforms were measured in response to a pure-tone and a speech stimulus and then analysed. It was hypothesised that the latency and amplitude measurements of children who had received a cochlear implant prior to 3.5 years of age would be the same as their chronologically age-matched, normal hearing peers. In addition, in order to assess whether the length of time in sound was the critical factor, or whether it was stimulation from a cochlear implant or hearing aids, a direct comparison between children using a cochlear implant versus hearing aids was made.

In general, we found that there were minimal differences in peak latency and amplitude measurements in waveforms when elicited by different types of acoustic stimuli. We also found minimal differences between the two children with hearing instruments matched for chronological age, suggesting that the auditory system does develop in a similar manner despite the type of hearing instrument used. Finally, the latency and amplitude measurements of children with cochlear implants were not conclusively aligned to either the normal hearing children matched for chronological age or those matched for "time-in-sound".

Therefore, we were unable to support either Sharma and colleagues' or Ponton and colleagues' research.

4.2 Differences in Peak Latencies and Amplitudes with Stimulus Type

4.2.1 Latency

The majority of children in the current study were found to have no statistically significant differences in their latency measurements when elicited by the pure-tone and the speech stimuli. This is consistent with previous literature which has documented that CAEP waveforms can be elicited using any type of acoustic stimuli, including speech, pure-tones, and clicks, and still result in comparable waveform morphology (Purdy et al., 2001; Sharma et al., 1997; Wunderlich & Cone-Wesson, 2006; Wunderlich et al., 2006).

The similarities in latency found in the present study is supported by Wunderlich and colleagues (2006) who examined the CAEP waveforms of 19 toddlers (13-41 months) and 20 children (4-6 years) recorded in response to a word, a high frequency pure-tone, and a low frequency pure-tone stimulus. Their study found that the P1, P2, and N2 peak latency measurements did not differ with stimulus type. However, N1 latency measurements were found to be longer when evoked by the low-frequency pure-tone stimulus than by words or high-frequency stimulus (Wunderlich et al., 2006). This difference may have been due to the differences in the stimuli frequencies, as the frequency of the low pure-tone was

400 Hz versus the 110 Hz of the fundamental frequency used in the speech stimulus. In contrast, the present study based the pure tone, 193 Hz, on the fundamental frequency of the /ba/ speech stimulus. This may explain the lack of any significant differences found in the majority of the N1 peak latencies, and the only child with a significant difference, CI 3, recording a longer N1 latency in response to the speech stimulus.

Nevertheless, statistically significant differences were found in the current study between the peak latency measurements recorded in response to the 193 Hz pure-tone stimulus and to the /ba/ speech stimulus for some children. Children CI 5 and TiS 4 recorded longer P1 latencies in response to the 193 Hz pure-tone stimulus, whereas the other two children, NH 3 and NH 4, recorded longer latencies in response to the /ba/ speech stimulus.

Longer P2 latencies were recorded in response to the 193 Hz pure-tone for four children, NH 1, NH 2, TiS 2, TiS 4, as well as the N2 latencies for CI 1, CI 5, NH 1, and NH 2. These differences in latency may have occurred due to the differences in the acoustic stimuli. Spectral information of the 193 Hz pure-tone is even throughout the duration, whereas a peak occurs at approximately 33 ms in the more spectrally complex /ba/ speech stimulus, as shown in Figure 12.

Therefore, firing in the cochlea and then throughout the auditory nervous system may have occurred at a slightly later stage in response to the speech stimulus.

4.2.2 Amplitude

Half of the children in the current study were found to have no significant differences in their P1 amplitude measurements recorded in response to the pure-tone and speech stimuli. The other children, only one of whom had a cochlear implant, were all found to have larger P1 amplitudes when elicited by the speech stimulus. Furthermore, the N1 amplitudes of only three children in the present study were found to significantly vary. CI 3 and NH 2 were found to have larger amplitude measurements when evoked by the speech stimulus, whereas CI 4 had larger N1 amplitudes recorded in response to the pure-tone.

Wunderlich and colleagues (2006) investigated CAEP waveforms of 19 toddlers and 20 children, recorded in response to a variety of stimuli, and found that there were no specific statistical differences in the P1 or N1 peak amplitudes in children, aged four to six years old, despite the type of stimulus used. This is consistent with half of the P1 amplitude results as well as with the majority of the N1 amplitude results found in the present study.

Almost half of the children recorded P2 amplitudes that were larger when elicited by the pure-tone stimulus, with only one child, CI 5, having P2 amplitude

measurements larger when evoked by the speech stimulus. The remaining children all had no significant differences in their P2 amplitudes. A previous study also found a stimulus-related effect on the P2 amplitude, where a low-frequency pure-tone evoked a larger amplitude response than either a high-frequency pure-tone or words, in children aged four to six years (Wunderlich et al., 2006).

Nine of the 16 children in the present study were found to have larger N2 amplitudes when recorded in response to the speech stimulus. In a study completed by Wunderlich and colleagues (2006), N2 was found to have larger peak amplitudes when elicited by either a low-frequency pure-tone or a speech stimulus as opposed to a high-frequency pure-tone stimulus in 19 toddlers (13-41 months) and 20 children (4-6 years). This is consistent with the almost half of the current findings, where a low-frequency pure-tone and the recorded speech stimulus both elicited similar N2 peak amplitudes.

Wunderlich and colleagues (2006) found that, while any auditory stimulus can effectively evoke CAEP waveforms, speech stimuli frequently elicited a larger response. They considered the possibility that this was due to speech being a spectrally complex stimulus as opposed to a pure-tone stimulus.

4.3 Latencies in Children with Cochlear Implants and Normal Hearing

The following sections 4.3.1 to 4.3.4 summarize the differences in latencies for each of the CAEP peaks between children with cochlear implants and those with normal hearing (matched for either chronological age or time in sound). These findings are then discussed with reference to the literature in Section 4.3.5.

4.3.1 P1 Latency

The P1 latency measurements recorded in response to the 193 Hz pure-tone stimulus in children CI 2 and CI 5 were found to not differ significantly from those measurements recorded by either of their corresponding normal hearing children. CI 1 and CI 4 were both found to have P1 latencies that were statistically different from their two corresponding normal hearing children when evoked in response to the pure-tone. The P1 latency measurements elicited by the 193 Hz pure-tone stimulus of CI 3 were statistically different from the latencies of the normal hearing child matched for "time-in-sound".

All but one child with a cochlear implant recorded no significant differences in P1 latencies from their two corresponding normal hearing children, when the CAEP was evoked using the /ba/ speech stimulus. CI 3 was again the only child to have significantly different latency measurements, but this time from the chronologically age-matched child with normal hearing.

4.3.2 N1 Latency

CAEPs evoked by the pure-tone resulted in children CI 1 and CI 2 having significantly different N1 latencies than their corresponding normal hearing children matched for "time-in-sound". The other three children were found to have no significant differences in N1 latency, evoked by the pure-tone, from either of their corresponding children with normal hearing.

Furthermore, no significant differences in N1 latency were found between any of the children when the CAEPs were elicited using a consonant-vowel /ba/ speech stimulus.

4.3.3 P2 Latency

In response to the 193 Hz pure-tone stimulus, children CI 1, CI 3, and CI 5 were all found to have P2 latency measurements significantly different from both of their corresponding normal hearing children. CI 2 was found to have a significantly different P2 latency from their chronologically age-matched normal hearing child, whereas CI 4 had P2 latency measurements significantly different from their normal hearing child matched for "time-in-sound".

There were no significant differences found between any of the children's P2 latency measurements when evoked by the recorded /ba/ speech stimulus.

4.3.4 N2 Latency

Children CI 1 and CI 4 were found to have significantly different N2 latency measurements, when evoked by the pure-tone, from their normal hearing child matched for "time-in-sound", whereas CI 2's N2 latency was significantly different from their chronologically age-matched normal hearing child. The other two children with cochlear implants were found to have inconclusive results with CI 3 recording no difference in N2 latency and CI 5 recording latency measurements significantly different from both normal hearing children.

CAEPs evoked by the /ba/ speech stimulus resulted in CI 2 and CI 3 having significantly different N2 latency measurements from their chronologically age-matched child with normal hearing. No differences were found between the N2 latencies of the other three children.

4.3.5 Summary of Latencies in Children with Cochlear Implants and Normal Hearing

The majority of the present study's results either found no significant difference between the latency measurements of the children with cochlear implants and their two corresponding normal hearing peers, or were significantly different from both children with normal hearing. Therefore, these findings are unable to support either Sharma and colleagues' literature that the CAEP waveforms of children with cochlear implants become equal to those of their age-matched peers

(Sharma, Dorman et al., 2005; Sharma, Dorman et al., 2002b) or Ponton and colleagues' research that the CAEP waveforms develop in a delayed manner reflecting the shorter time children with cochlear implants have actually been able to hear for (Ponton, Don, Eggermont, Waring, Kwong et al., 1996; Ponton, Don, Eggermont, Waring, & Masuda, 1996).

The majority of the latency measurements recorded in response to the 193 Hz pure-tone stimulus and to the /ba/ speech stimulus in children with cochlear implants were found to not differ significantly from those measurements recorded in the chronologically age matched normal-hearing children. This finding does not contradict Sharma and colleagues' literature that the CAEP waveforms of children with cochlear implants become equal to those of their age-matched peers (Sharma, Dorman et al., 2005; Sharma, Dorman et al., 2002b).

Sharma and colleagues (2002) recorded CAEPs from 136 participants with normal hearing, aged from one month to 20 years old, and 107 participants with cochlear implants, aged from two to 35 years old. All CAEPs were evoked using a synthesised speech stimulus /ba/. They found that 55 out of the 57 children who received their cochlear implant prior to 3.5 years of age had P1 latencies within the same range as their chronologically age-matched peers with normal hearing. In contrast, only one out of 21 participants who received their cochlear implant after seven years of age fell within the appropriate range (Sharma et al., 2002). This finding was supported by an additional study which found no significant

difference in the P1 latency measurements of 18 children who received cochlear implants prior to 3.5 years of age and 18 chronologically age-matched children with normal hearing (Sharma et al., 2002).

Furthermore, in the present study, no statistically significant difference was found in the majority of latency measurements between the children with cochlear implants and the normal hearing children matched for "time-in-sound" either. This, therefore, does not contradict Ponton and colleagues' research either, that the CAEP waveforms develop in a delayed manner reflecting the shorter time children with cochlear implants have actually been able to hear for (Ponton, Don, Eggermont, Waring, Kwong et al., 1996; Ponton, Don, Eggermont, Waring, & Masuda, 1996).

Ponton and colleagues (1996) investigated the P1 latencies of 31 children with normal hearing and 12 children with cochlear implants using click stimuli. They found that the P1 latency in children with cochlear implants developed at a normal rate but was approximately delayed by the duration of deafness the child had experienced (Ponton et al., 1996). This finding was further supported in a study by Ponton and colleagues (1997), which again emphasised that once adequate auditory stimulation is provided, the central auditory pathway continues to develop at a normal rate but delayed by the time of deafness.

Overall, the latency measurements obtained in this current study are unable to support either of these current theories due to the absence of statistical differences found between the children with cochlear implants and one of the two groups of children with normal hearing. The lack of statistical findings in the present study is most likely due to the limited sample size of children with cochlear implants that were available to participate as well as the large amount of variability in the results from subjects. Furthermore, the majority of the ages of the normal hearing children in the two different groups overlapped, and often no significant differences were found between the normal hearing children from the two groups when comparing the latencies recorded in response to either the pure-tone or the speech stimulus.

When compared to normative data reported in the literature, four of the children with cochlear implants (CI 1, CI 2, CI 3, and CI 4) have P1 latencies within the appropriate range for their age (Sharma et al., 1997; Wunderlich et al., 2006) when elicited by the 193 Hz pure-tone stimulus. The P1 latency of the other child with cochlear implants is slightly longer than the range for their age as well as for the normative range based on their "time-in-sound" age (Wunderlich et al., 2006). CAEPs evoked by the /ba/ speech stimulus also resulted in four of the children with cochlear implants (CI 1, CI 3, CI 4, and CI 5) having P1 latencies within the appropriate range for their age (Sharma et al., 1997; Wunderlich et al., 2006). CI 2 had P1 latency measurements that only just overlapped with the normative range matched for chronological age. The mean P1 latency measurement was well

within the normative range based on their "time-in-sound" age (Sharma et al., 1997; Wunderlich et al., 2006).

4.4 Amplitudes in Children with Cochlear Implants and Normal Hearing

The following sections 4.4.1 to 4.4.4 summarize the differences in amplitudes for each of the CAEP peaks between children with cochlear implants and those with normal hearing (matched for either chronological age or time in sound). These findings are then discussed with reference to the literature in Section 4.4.5.

4.4.1 P1 Amplitude

Children with cochlear implants were found to have either no significant differences (CI 1 and CI 5) from the normal hearing peers or significant differences (CI 2, CI 3, and CI 4) from both their corresponding normal hearing children in P1 amplitude when evoked by the 193 Hz pure-tone.

In a similar manner, when the CAEPs were evoked by the /ba/ speech stimulus, CI 4 had significantly different P1 amplitude measurements from both matched normal hearing children, while the remaining children with cochlear implants were found to have no significant differences from their normal hearing peers.

4.4.2 N1 Amplitude

The N1 amplitude evoked by the pure-tone for CI 2 was found to be significantly different from their chronologically age-matched peer with normal hearing. Children CI 1 and CI 5 were found to have N1 amplitudes that were not

statistically different from either of their corresponding normal hearing children, while CI 3 and CI 4 had N1 amplitudes that were significantly different from both normal hearing peers.

Three children, CI 1, CI 2, and CI 3, recorded N1 amplitudes in response to the /ba/ speech stimulus that were not significantly different from either of their corresponding normal hearing children. The N1 amplitude measurements of CI 4 were significantly different from both matched children with normal hearing. CI 5 had N1 amplitude measurements significantly different from their chronologically age-matched peer with normal hearing, when elicited by the /ba/ speech stimulus.

4.4.3 P2 Amplitude

Three children with cochlear implants, CI 1, CI 3, and CI 5, were found to have P2 amplitudes, elicited by the pure-tone stimulus, that were not significantly different from either of their normal hearing peers. CI 2 was found to have a P2 amplitude that was significantly different from the normal hearing child matched for chronological age, whereas CI 4's amplitude was significantly different from the normal hearing child matched for "time-in-sound".

Three children were found to have inconclusive results when P2 amplitude was evoked by the /ba/ speech stimulus. CI 3 had amplitude measurements that were not significantly different from either normal hearing peer, while CI 4 and CI 5 had amplitudes that were different from both their matched children with normal

hearing. The P2 amplitude recorded by CI 1 was significantly different from the chronologically age-matched child with normal hearing. In contrast, CI 2 recorded a P2 amplitude measurement that was significantly different from the normal hearing child matched for "time-in-sound".

4.4.4 N2 Amplitude

CI 4 recorded an N2 amplitude measurement that was significantly different from both normal hearing peers, when evoked by a pure-tone stimulus. The remaining four children with cochlear implants all had no significant differences in the N2 amplitude measurements when compared to their corresponding normal hearing children.

In a similar manner, when evoked by the speech stimulus, CI 1, CI 4, and CI 5 all had significantly different N2 amplitudes from their normal hearing children, while CI 2 and CI 3 recorded no significant differences from their corresponding peers.

4.4.5 Summary of Amplitudes in Children with Cochlear Implants and Normal Hearing

The majority of the peak amplitude measurements of the individual children with cochlear implants were found to have either no significant difference from the children with normal hearing or to be significantly different from both groups of

normal hearing peers. This remained the case whether the waveforms were evoked by the 193 Hz pure-tone or the recorded speech stimulus.

Only five peak amplitude measurements were revealed to have significant differences from the normal hearing child matched for chronological age. These results do not contradict Ponton and colleagues theory that children with cochlear implants have measurements similar to their normal hearing peers who are matched for "time-in-sound". However, two peak amplitude results were found to differ significantly from the child matched for "time-in-sound" and, therefore, we are also unable to contradict Sharma and colleagues supposition that children who receive cochlear implants prior to 3.5 years of age are able to attain the same measurements as their chronologically age-matched normal hearing peers.

As was the case for the latency measurements, when all of these results were taken together, the present study was unable to conclusively demonstrate that amplitude measurements of children with cochlear implants were equivalent to either chronologically age matched normal hearing children or to children matched for time-in-sound.

When compared to normative data published in the literature, only two children with cochlear implants had P1 amplitude measurements within the appropriate range for their chronological age. CI 2 recorded a mean P1 amplitude within the normative data for their age when in response to the speech stimulus. CI 5 also

had age appropriate P1 amplitudes recorded in response to both the pure-tone and the speech stimuli (Sharma et al., 1997). The other three children with cochlear implants had P1 amplitude measurements greater than those published for their chronological age ranges (Sharma et al., 1997; Wunderlich et al., 2006). However, it must be emphasized that amplitude measurements of CAEP waveforms are more variable than latency measurements and are, therefore, less reliable when comparing data, especially between studies.

4.5 Latencies and Amplitudes in Children with Cochlear Implants and Hearing Aids

The findings of the present study demonstrated no significant differences between any of the latency measurements recorded for the child with a cochlear implant and the child with hearing aids when evoked by either the pure-tone or the speech stimuli. This supports the hypothesis that the evoked responses in age matched children with a hearing impairment, who received a hearing instrument before 3.5 years of age, would be identical, whether they are receiving amplification from a cochlear implant or a hearing aid. This strengthens the findings that children's auditory systems develop comparatively so long as they are receiving appropriate amplification, whether this be via a cochlear implant or through the use of hearing aids. However, future research on a larger scale is needed to support this emerging theory suggested by this preliminary case study comparing the CAEP waveforms of children using different types of hearing instruments.

In contrast, differences were found in the amplitude measurements of the two age-matched children with a cochlear implant and with hearing aids. The P1 amplitude measurements of CI 4 and HA 4 were found to be significantly different when elicited by the 193 Hz pure-tone. The two children's P1, N1, and N2 amplitude measurements were significantly different when evoked by the /ba/ speech stimulus. These results reflect the high intra- and inter-subject variability that can occur in CAEP amplitude measurements (Hall, 1991).

4.6 Clinical Implications

The majority of current research has focused on investigating P1 and has demonstrated that there are clear changes in latency measurements once a previously deprived auditory system receives auditory stimulation via a cochlear implant and/or hearing aids (Eggermont et al., 1997; Ponton et al., 1996; Sharma et al., 1997; Sharma et al., 2002b). Decreases in P1 latency can provide an objective measurement that a child is receiving adequate amplification from their hearing instrument. Therefore, this would assist in the difficult decision making process of whether to implant in difficult to test populations, such as infants. If latency measurements do not decrease to within the age-appropriate range during the hearing aid trial, then this would suggest they are not receiving sufficient stimulation and a cochlear implant should be considered (Sharma, Dorman, Spahr et al., 2002; Sharma, Dorman et al., 2002a; Sharma, Dorman et al., 2002b).

Adequate auditory stimulation is a necessary precursor to the development of speech and language. Therefore, it follows that once a child receives appropriate hearing instruments for their needs there should be a follow on affect in the child's speech and language development (Sharma et al., 2004). A preliminary study investigated two infants, who received cochlear implants at 13 and 14 months of age. P1 latency was measured as was language development via the children's use of pre-canonical and canonical babbling (Sharma et al., 2004). They found that decreases in latency were also reflected by increases in canonical babbling. This suggests that early communicative behaviours may follow a similar developmental path as P1 latencies, and is, thus, also improved by adequate stimulation of the auditory nervous system (Sharma et al., 2004). Therefore, chronological age-appropriate P1 latency measurements may be a strong indicator that the auditory system is working adequately for normal speech and language development.

4.7 Limitations of the current study

The two main limitations of this study related to the level of noise in the electrophysiological waveforms and the difficulty in obtaining research participants.

Despite low electrode impedances being achieved in every case, some of the younger subjects produced large amounts of noise due to movement, resulting in a high rejection-rate and subsequent reduction in waveform quality.

In the planning stages for the current study, it was anticipated that 16 cochlear implant participants would be recruited, and that these subjects would be age matched to normally-hearing peers on the basis of time-in-sound and chronological age. Of the invitations that were sent to prospective participants, there were only seven replies. Of those seven, five were willing to participate. This reduction in sample size made it difficult to achieve the levels of statistical significance required for a study of this type. One solution to this problem may be to conduct research of this nature using a larger pool of cochlear implantees, perhaps drawn from an overseas population.

4.8 Future Research

It is important that future research further investigates the CAEP waveform morphology of children with cochlear implants and determines whether the latency and amplitude measurements are equal to chronologically age matched normal hearing peers, as stated by Sharma and colleagues or whether they are equivalent to normal hearing children matched for time-in-sound, as maintained by Ponton and colleagues. Such a study needs to be done on a large scale in order for significant results to be clearly illustrated.

In addition, it would be valuable for future research to investigate the similarities and/or differences in CAEP waveform morphology in children with cochlear implants and with hearing aids. This will aid understanding of how these different hearing instruments work to stimulate and promote the maturation of the auditory system in children.

Future research investigating the manner in which CAEP waveforms change and mature in individual children would also be beneficial. In particular, a longitudinal study recording CAEP waveforms in children when they are initially identified with a profound hearing loss, during the hearing aid trial, and then on multiple occasions once they receive a cochlear implant. This would assist in developing a thorough understanding of the manner in which this simple, objective measurement may be used to support the process of cochlear implantation in children.

4.9 Summary and Conclusions

The present study found that the majority of latency and amplitude measurements are the same in children with hearing instruments and in children with normal hearing despite the type of acoustic stimuli used to elicit the CAEP waveform.

The findings reported in the current study were unable to conclude whether the latency and amplitude measurements of children with cochlear implants more closely resembled those of chronologically age matched normal hearing children or children matched for time-in-sound. This is most likely because of the small number of participants involved in this study, and therefore, future research investigating a larger number of children is recommended.

Finally, the current research found no significant differences in latency and minimal differences in amplitude measurements for the child with a cochlear implant and the child with hearing aids. This provides support for the hypothesis that children who receive appropriate amplification prior to 3.5 years of age will have comparable waveforms despite the type of hearing instrument used.

Although it remains inconclusive whether the CAEP waveforms in children who receive cochlear implants prior to 3.5 years of age are able to catch-up and become age-appropriate, it is unmistakable that this remains an important question. In particular, this information would provide further support for early implantation in children with severe to profound hearing losses.

References

Burkard, R. F. & Secor, C. (2002). In Katz, J. (Ed.). *Handbook of Clinical Audiology* (5th ed.). Lippincott Williams & Wilkins: Philadelphia, PA.

Calhoun, B. (2008). *The Anatomy of Hearing*. Retrieved January 22, 2008 from http://www.brainconnection.com/topics/?main=anat/auditory-anat

Caton, R. (1875). The electrical currents of the brain. *Br Med J, 2,* 278.

Danmeter (2006). *The AEP Monitor/2*. Retrieved January 22, 2008 from http://www.danmeter.dk/products/neuromonitoring/aepmonitor2/index.html

Davis, P. A. (1939). Effects of acoustic stimuli on the working human brain. *J Neurophysiol, 2,* 494-499.

Eggermont, J. J., Ponton, C. W., Don, M., Waring, M. D., & Kwong, B. (1997). Maturational delays in cortical evoked potentials in cochlear implant users. *Acta Otolaryngol, 117*(2), 161-163.

Gilley, P. M., Sharma, A., Dorman, M., & Martin, K. (2005). Developmental changes in refractoriness of the cortical auditory evoked potential. *Clin Neurophysiol, 116*(3), 648-657.

Gross, A. (2003). *Fitting Techniques for the Pediatric Cochlear Implant Patient.* Retrieved January 24, 2008 from http://www.audiologyonline.com/articles/pf_article_detail.asp?article_id=424

Hall, J. W. (1991). *Handbook of Auditory Evoked Responses.* Allyn and Bacon: Boston.

Kelly, A. S., Purdy, S. C., & Thorne, P. R. (2005). Electrophysiological and speech perception measures of auditory processing in experienced adult cochlear implant users. *Clin Neurophysiol, 116*(6), 1235-1246.

McArthur, G. M., Bishop, D. V. M., & Proudfoot, M. (2003). Do video sounds interfere with auditory event-related potentials? *Behaviour Research Methods, Instruments, & Computers, 35(2), 329-333.*

McPherson, D. L. & Ballachanda, B. (2000). Middle and long latency auditory evoked potentials. In Roeser, R. J., Valente, M., & Hosford-Dunn, H. (Eds.). *Audiology Diagnosis*. Thieme Medical Publishers, Inc: New York.

Musiek, F. E. & Oxholm, V. B. (2000). Anatomy and physiology of the central auditory nervous system: A clinical perspective. In Roeser, R. J., Valente, M., & Hosford-Dunn, H. (Eds.). *Audiology Diagnosis*. Thieme Medical Publishers, Inc: New York.

Ponton, C. W., & Don, M. (2003). Cortical auditory evoked potentials recorded from cochlear implant users: methods and applications. In H. E. Cullington (Ed.), *Cochlear Implants: Objective Measures* (pp. 187-230). London: Whurr Publishers Ltd.

Ponton, C. W., Don, M., Eggermont, J. J., Waring, M. D., Kwong, B., & Masuda, A. (1996). Auditory system plasticity in children after long periods of complete deafness. *Neuroreport, 8*(1), 61-65.

Ponton, C. W., Don, M., Eggermont, J. J., Waring, M. D., & Masuda, A. (1996). Maturation of human cortical auditory function: differences between normal-hearing children and children with cochlear implants. *Ear Hear, 17*(5), 430-437.

Purdy, S. C., Kelly, A. S., & Thorne, P. R. (2001). Auditory evoked potentials as measures of plasticity in humans. *Audiol Neurootol, 6*(4), 211-215.

Rappaport, J. M. & Provençal, C. (2002). Neuro-otology for audiologists. In Katz, J. (Ed.). *Handbook of Clinical Audiology* (5th ed.). Lippincott Williams & Wilkins: Philadelphia, PA.

Sharma, A., Dorman, M., Spahr, A., & Todd, N. W. (2002). Early cochlear implantation in children allows normal development of central auditory pathways. *Ann Otol Rhinol Laryngol Suppl, 189*, 38-41.

Sharma, A., & Dorman, M. F. (2006). Central auditory development in children with cochlear implants: clinical implications. *Adv Otorhinolaryngol, 64*, 66-88.

Sharma, A., Dorman, M. F., & Kral, A. (2005). The influence of a sensitive period on central auditory development in children with unilateral and bilateral cochlear implants. *Hear Res, 203*(1-2), 134-143.

Sharma, A., Dorman, M. F., & Spahr, A. J. (2002a). Rapid development of cortical auditory evoked potentials after early cochlear implantation. *Neuroreport, 13*(10), 1365-1368.

Sharma, A., Dorman, M. F., & Spahr, A. J. (2002b). A sensitive period for the development of the central auditory system in children with cochlear implants: implications for age of implantation. *Ear Hear, 23*(6), 532-539.

Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology, 104*, 540-545.

Sharma, A., Martin, K., Roland, P., Bauer, P., Sweeney, M. H., Gilley, P., et al. (2005). P1 latency as a biomarker for central auditory development in children with hearing impairment. *J Am Acad Audiol, 16*(8), 564-573.

Sharma, A., Tobey, E., Dorman, M., Bharadwaj, S., Martin, K., Gilley, P., & Kunkel, F. (2004). Central auditory maturation and babbling development in infants with cochlear implants. *Arch Otolaryngol Head Neck Surg, 130,* 511-516.

Stapells, D. R. (2002). Cortical event-related potentials to auditory stimuli. In Katz, J. (Ed.). *Handbook of Clinical Audiology* (5th ed.). Lippincott Williams & Wilkins: Philadelphia, PA.

Wunderlich, J. L., & Cone-Wesson, B. K. (2006). Maturation of CAEP in infants and children: A review. *Hearing Research, 212*(1-2), 212-223.

Wunderlich, J. L., Cone-Wesson, B. K., & Shepherd, R. (2006). Maturation of the cortical auditory evoked potential in infants and young children. *Hearing Research, 212*(1-2), 185-202.

Zwolan, T. (2002). Cochlear implants. In Katz, J. (Ed.). *Handbook of Clinical Audiology* (5th ed.). Lippincott Williams & Wilkins: Philadelphia, PA.

Appendices

Appendix 1

Upper South A Regional Ethics Committee Approval

Health & Disability Ethics Committees

17 July 2007

Jennifer Walker
46 Hudson Street
Bryndwr
Christchurch

Dear Jennifer Walker,

Maturation of cortical auditory evoked potentials in children with normal hearing and hearing impairment
Investigators: J Walker, Dr G O'Beirne (Supervisor), Dr V Looi
Ethics ref: URA/07/04/032

The above study has been given ethical approval by the **Upper South A** Ethics Committee.

Approved Documents
Information sheet and consent form version.

Accreditation
The Committee involved in the approval of this study is accredited by the Health Research Council and is constituted and operates in accordance with the Operational Standard for Ethics Committees, April 2006.

Final Report
The study is approved until **31 July 2008**. A final report is required at the end of the study and a form to assist with this is available from the Administrator. If the study will not be completed as advised, please forward a progress report and an application for extension of ethical approval one month before the above date. Report forms are available from the administrator.

Amendments
It is also a condition of approval that the Committee is advised of any adverse events, if the study does not commence, or the study is altered in any way, including all documentation eg advertisements, letters to prospective participants.

Please quote the above ethics committee reference number in all correspondence.

It should be noted that Ethics Committee approval does not imply any resource commitment or administrative facilitation by any healthcare provider within whose facility the research is to be carried out. Where applicable, authority for this must be obtained separately from the appropriate manager within the organisation.

We wish you well with your study.

Yours sincerely

Alieke Dierckx
Upper South A Ethics Committee Administrator
Email: alieke_dierckx@moh.govt.nz

Appendix 2

University of Canterbury Human Ethics Committee Approval

HEC Ref: 2007/34

6 March 20097

Ms Jennifer Walker
Communication Disorders
UNIVERSITY OF CANTERBURY

Dear Jennifer

The Human Ethics Committee advises that your research proposal "Maturation of cortical auditory evoked potentials in children with normal hearing and hearing impairment" has been considered and approved. However this approval is subject to the amendments you have outlined in the additional papers provided.

Yours sincerely

Dr Michael Grimshaw
Chair, Human Ethics Committee

Appendix 3

Information Sheet

1 June 2007

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

INFORMATION SHEET

Title: **The way sound-evoked brain activity matures in children with normal hearing and children with hearing impairment**

Principal Investigator:	Jenny Walker
Position:	Master of Audiology student
Address:	University of Canterbury Communication Disorders department Creyke Road, Christchurch
Phone Numbers:	0800 864-837 (select option 2) 027 286-1015
E-mail:	jkn21@student.canterbury.ac.nz
Supervisor:	Dr Greg O'Beirne
Position:	Lecturer in Audiology
Address:	University of Canterbury Communication Disorders department Creyke Road, Christchurch
Phone Number:	0800 864-837 (select option 2) 03 364-2987 ext: 7085
E-mail:	gregory.obeirne@canterbury.ac.nz

Dear Parent

You and your child are invited to take part in a student research study at the University of Canterbury Speech and Hearing Clinic. This study will be looking at the brain activity that occurs in response to sound in children with

cochlear implants, hearing aids, and normal hearing. The aim of the study is to determine how this brain activity changes in children with cochlear implants.

We are hoping to involve 16 children with cochlear implants, 16 children with hearing aids, and 32 children with normal hearing.

The research will be carried out at the University of Canterbury Speech and Hearing Clinic on Creyke Road, Ilam, Christchurch.

You and your child will need to attend one session that will last approximately 60 minutes. Appointments can be booked at a time that is most suitable for you, including during the day, after school, in the weekends, or during the school holidays.

If your child does not wear a cochlear implant or hearing aids, then prior to completing the study, your child's hearing will be screened to ensure that their hearing is within normal limits.

During the appointment your child will be seated comfortably and allowed to watch a movie of their own choice. Five button contacts will be placed on your child's head. This does not hurt (see risks below), and is like placing stickers that are attached to wires onto the head. These wires are hooked up to the computer, which can then record tiny brainwaves that occur in response to sounds that will be played through a loud-speaker in the room. Your child is not required to attend to the sounds coming out of the loud-speaker or to respond to them in anyway. Once this is completed, the contacts will be gently removed and the results will be stored in the computer, so that they can be analysed at a later date.

What are the benefits of the study?

- a free hearing screening test for children assumed to have normal hearing
- Benefits will also accrue for other children in the future – this is not a direct benefit for you or your child, but results from this study can help us to understand the optimal time for children to receive a cochlear implant, as well as providing us with an understanding of the way in which their auditory pathway develops once they receive appropriate stimulation from their cochlear implant.

What are the risks of the study?

- There is a possibility that the hearing screening test will indicate your child has a hearing loss that you were not previously aware of. In this case we would provide further testing, advice, and if necessary referral to other specialist services
- The electrode preparation involves scrubbing of the scalp and may cause some minor redness

Inclusion Criteria

- Children with cochlear implants who received their cochlear implant prior to 3.5 years of age
- Children with hearing aids who received their hearing aid prior to 3.5 years of age, and who has had it for the same length of time as one of the corresponding children with cochlear implants
- Children with normal hearing who are the same age as the corresponding children with cochlear implants
- Children with normal hearing who are the same age as length of time the corresponding children have had their cochlear implants for (i.e. a child who was had a cochlear implant for 2 years will be matched with a 2-year old child with normal hearing)

You and your child's participation are entirely voluntary. Your child does not have to take part in this study, and if you and/or your child choose not to take part this will not affect any future treatment or care from any providers.

If you do agree to take part you and/or your child are free to withdraw from the study at any time, without having to give a reason and this will in no way affect your future treatment or care from any providers.

You can get more information about the study, or discuss it further, by contacting the principal investigator, Jenny Walker, or the supervisor, Greg O'Beirne.

You may have a friend, family, or whanau support to help you understand the risks and/or benefits of the study and any other explanation you may require.

If you have any queries or concerns regarding your rights as a participant in this study you may wish to contact an independent Health and Disability Advocate:
Free Phone in the South Island: 0800 377-766
Free Fax (New Zealand wide): 0800 2787-7678 (0800 2 SUPPORT)
E-mail (New Zealand wide): advocacy@hdc.org.nz

No material which could personally identify your child will be used in any reports on this study. Each child will be assigned a number or letter for classification purposes.

Records will be kept in a locked cabinet in a lockable office at the University of Canterbury Communication Disorders department. Some data will also be stored on password-protected computers. Only those researchers directly involved in the research will have access to the recorded data.

If you would like to receive a copy of your child's results, please let us know. Otherwise, the research results will hopefully be published in appropriate journals and/or presented at conferences. There will be a delay between the time that the data is recorded and when the study is published.

This study has received ethical approval from the Upper South A Regional Ethics Committee and University of Canterbury Ethics Committee.

If you have any further questions about this study, please feel free to contact us.

Yours sincerely

Jenny Walker

Appendix 4

Consent Form

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

CONSENT FORM FOR PARENT/CAREGIVER/WHANAU

Title: The way sound-evoked brain activity matures in children with normal hearing and children with hearing impairment

Principal Investigator: Jenny Walker

Participant's Name: _____

I have read and I understand the information sheet dated 1 June 2007 for people taking part in the student study designed to record their sound-evoked brainwaves. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study.

I understand that taking part in this study is voluntary and that I or my child may withdraw from the study at any time if I or he/she wishes, and this will not affect any future care or assistance from any of my usual health providers.

I understand that his/her participation in this study is confidential and that no material which could identify him/her will be used in any reports on this study.

I have had time to consider whether to take part.

I know whom to contact if I or my child would like to withdraw from the study.

This study has been given ethical approval by the Upper South A Regional Ethics Committee and University of Canterbury Ethics Committee. This means that the Committees may check at any time that the study is following appropriate ethical procedures.

I/my child would like a copy of my child's results

YES/NO

I/my child would like his/her GP to be informed of
his/her participation in and a copy of my child's results

YES/NO

I/my child would like his/her Audiologist to be informed of
his/her participation in and a copy of my child's results

YES/NO

I give consent for Jenny Walker to approach the Southern Cochlear Implant Programme / Advisor of Deaf Children / Audiologist to access my child's records for the purposes of this study

YES/NO

Signed: _____ Date: _____

Printed Name: _____

Relationship to Participant: _____

Contact Phone Numbers: _____

Contact E-mail Address: _____

Address for results: _____

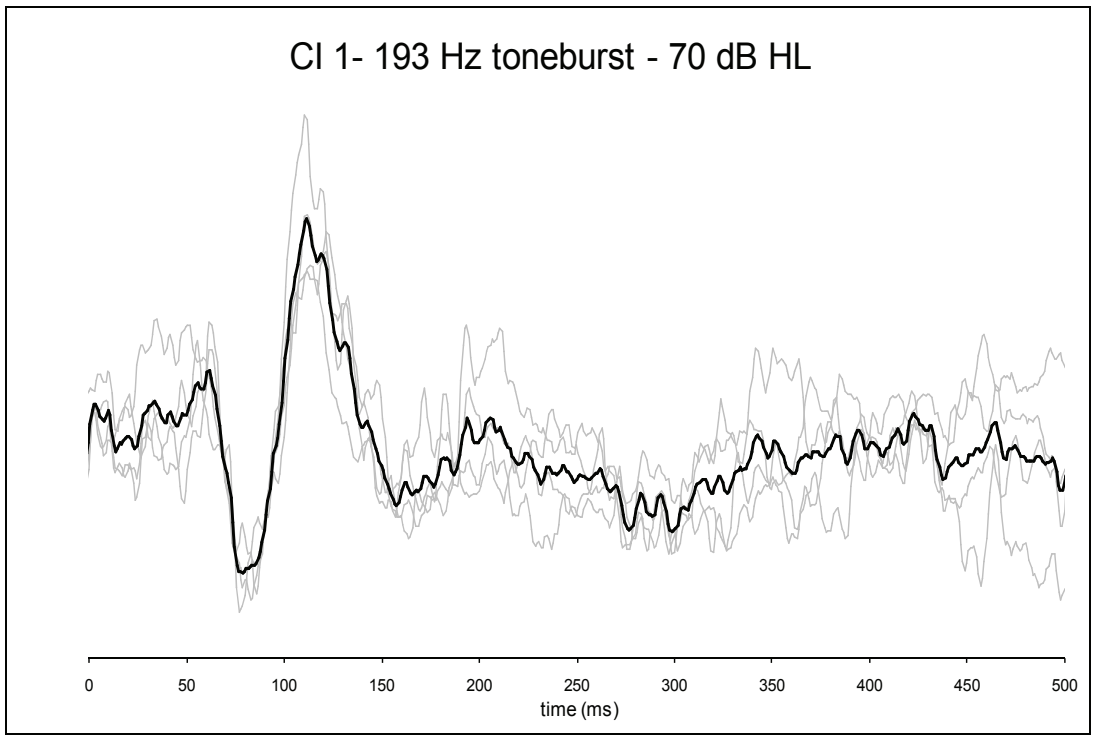
_____

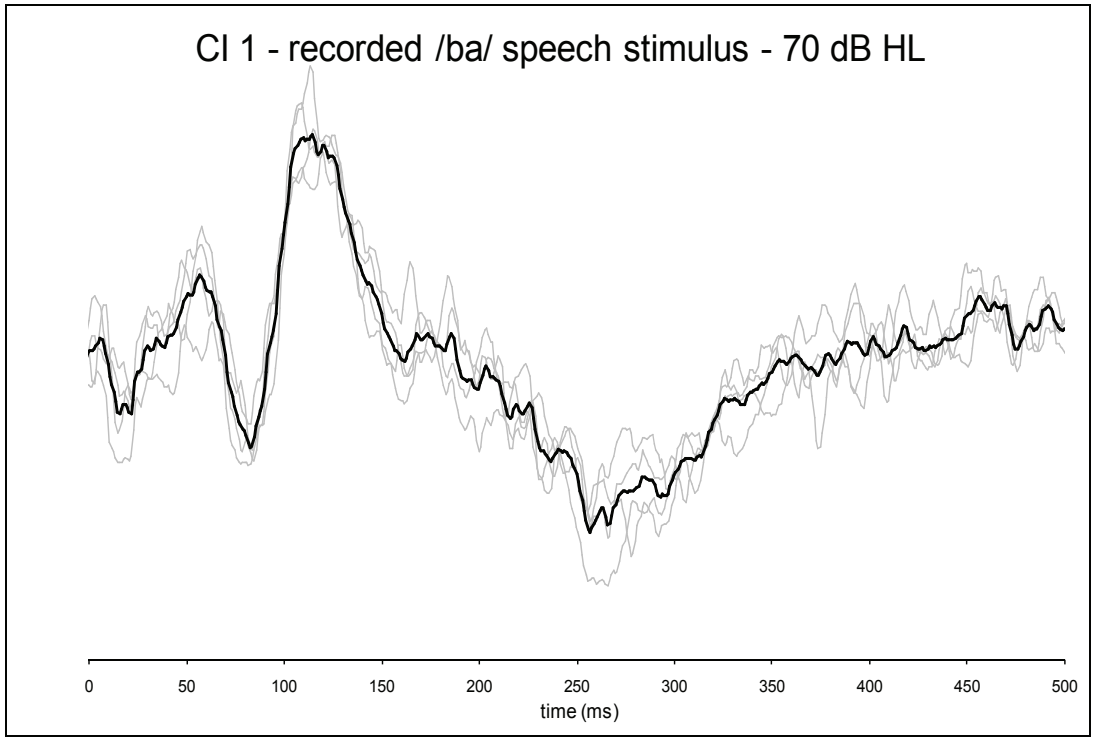
CONTACT DETAILS

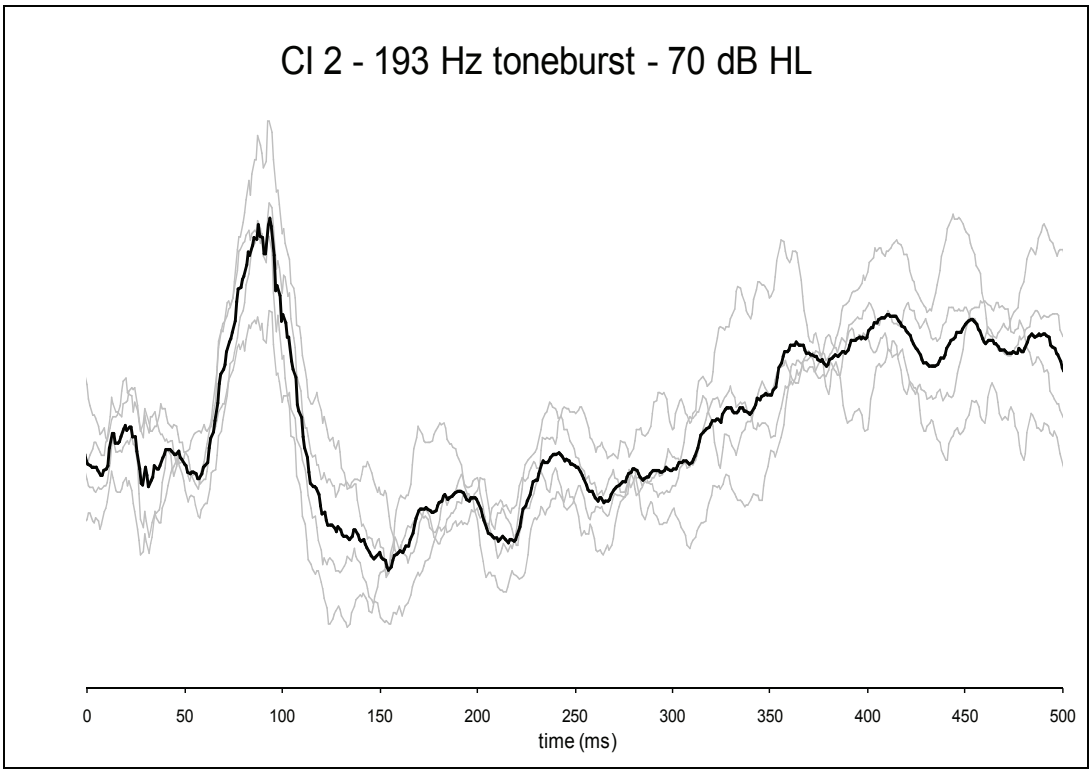
Researcher: Jenny Walker
Position: Master of Audiology student
Contact Number: 0800 864-837 (option 2)
Cell-phone Number: 027 286-1015
E-mail: jkn21@student.canterbury.ac.nz

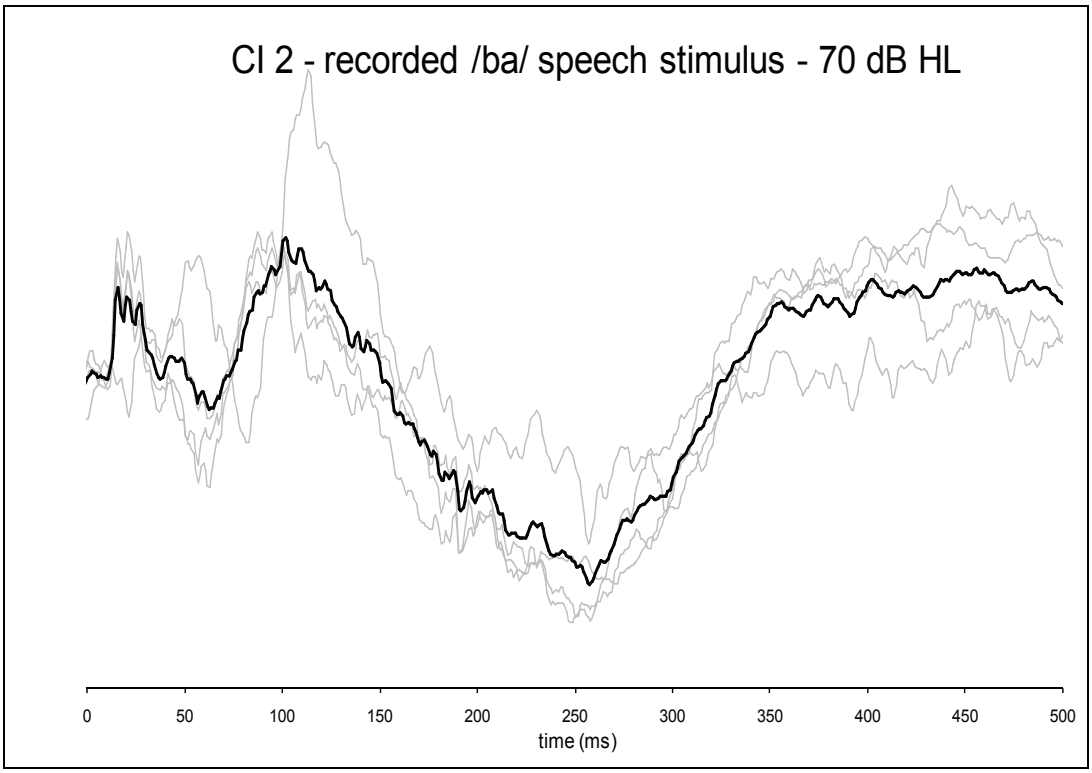
Supervisor: Dr Greg O'Beirne
Position: Lecturer in Audiology
Contact Number: 0800 864-837 (option 2)
Contact Number: 03 364-2987 ext 7085
E-mail: gregory.obeirne@canterbury.ac.nz

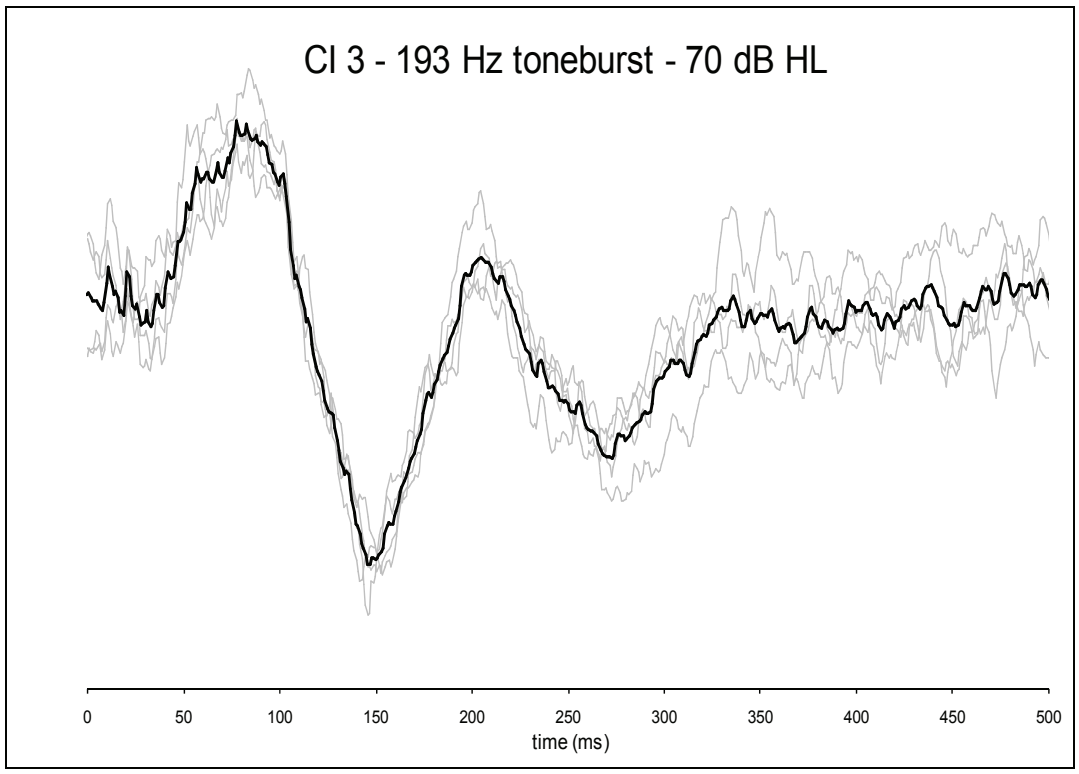
Appendix 5
Raw and Totalled Waveforms

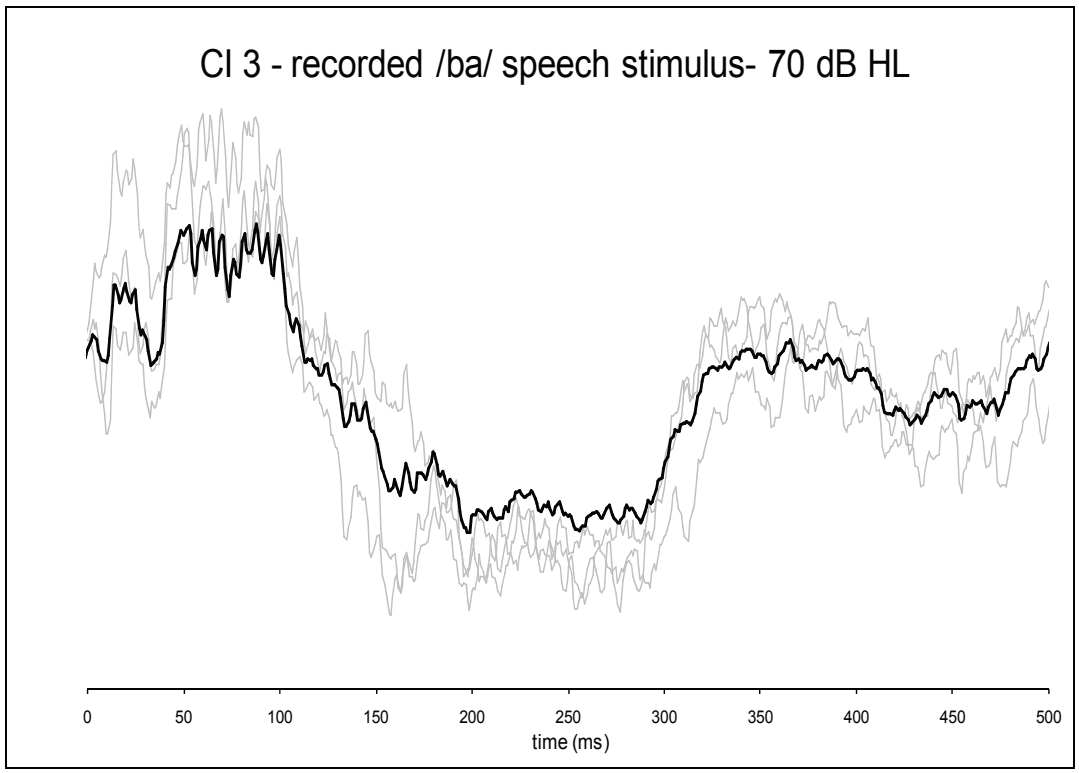


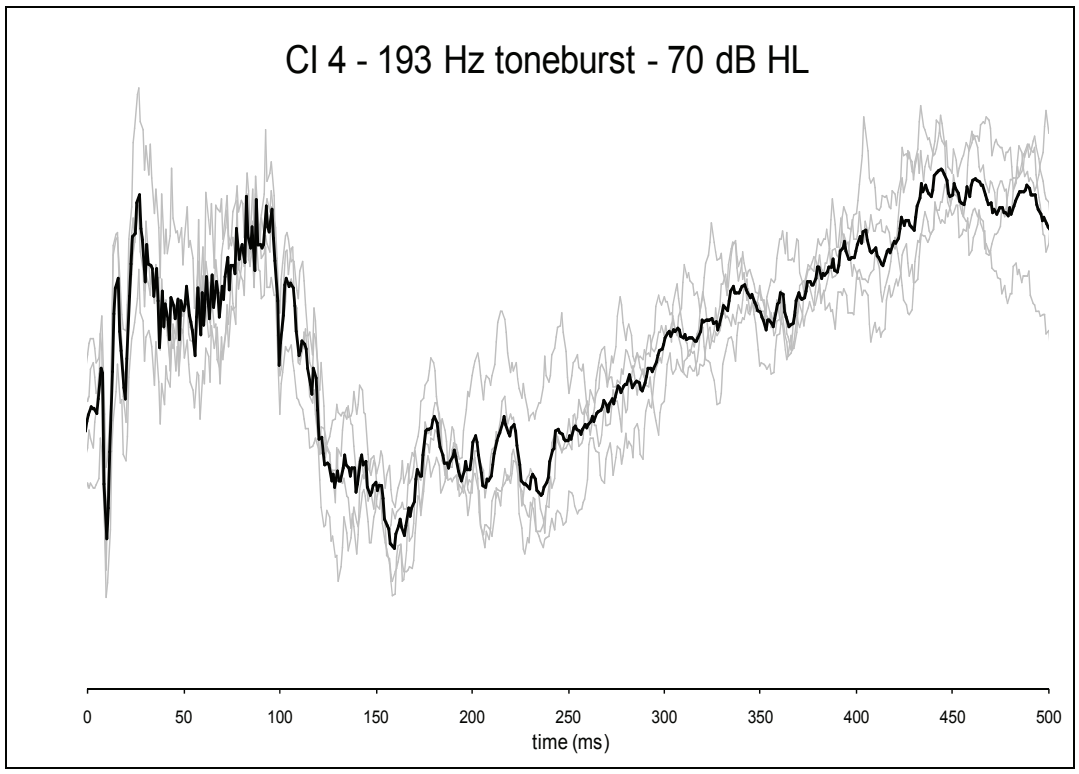


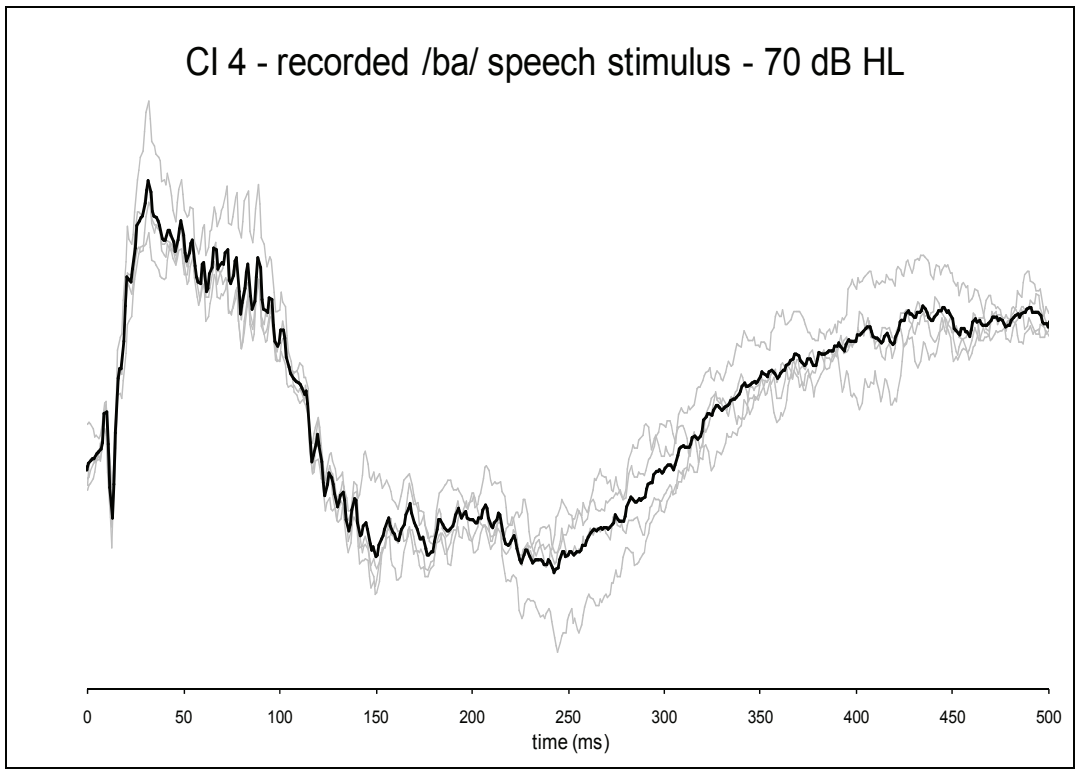

CI 2 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

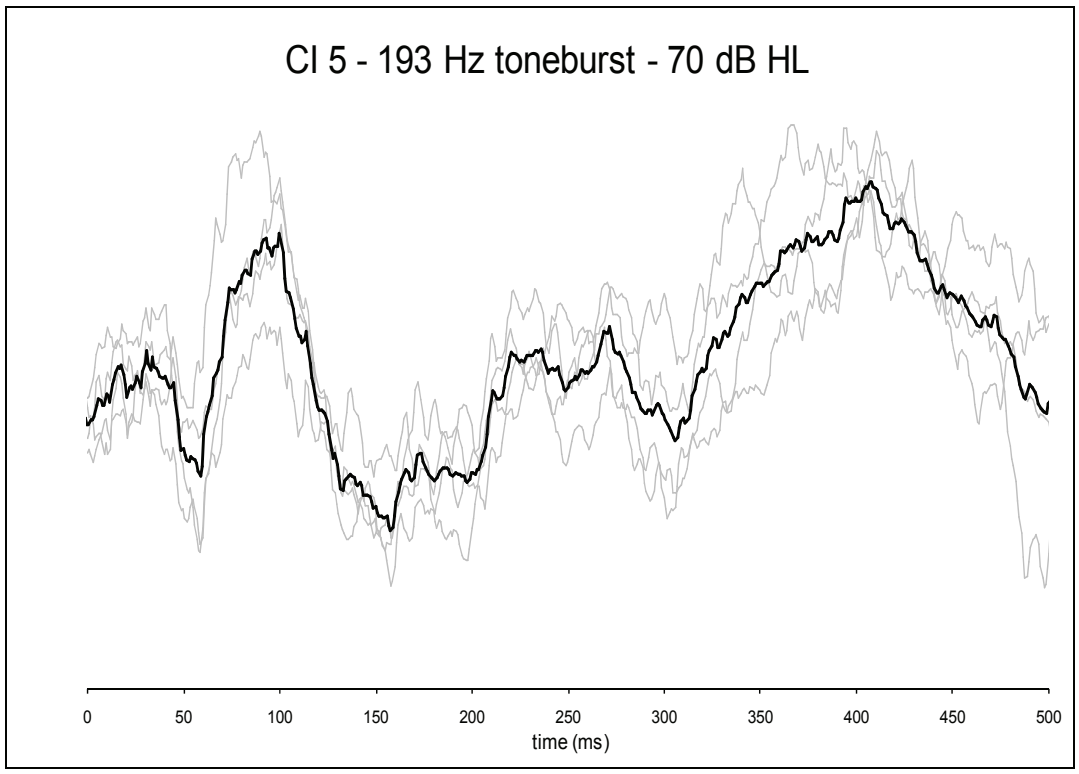

CI 2 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

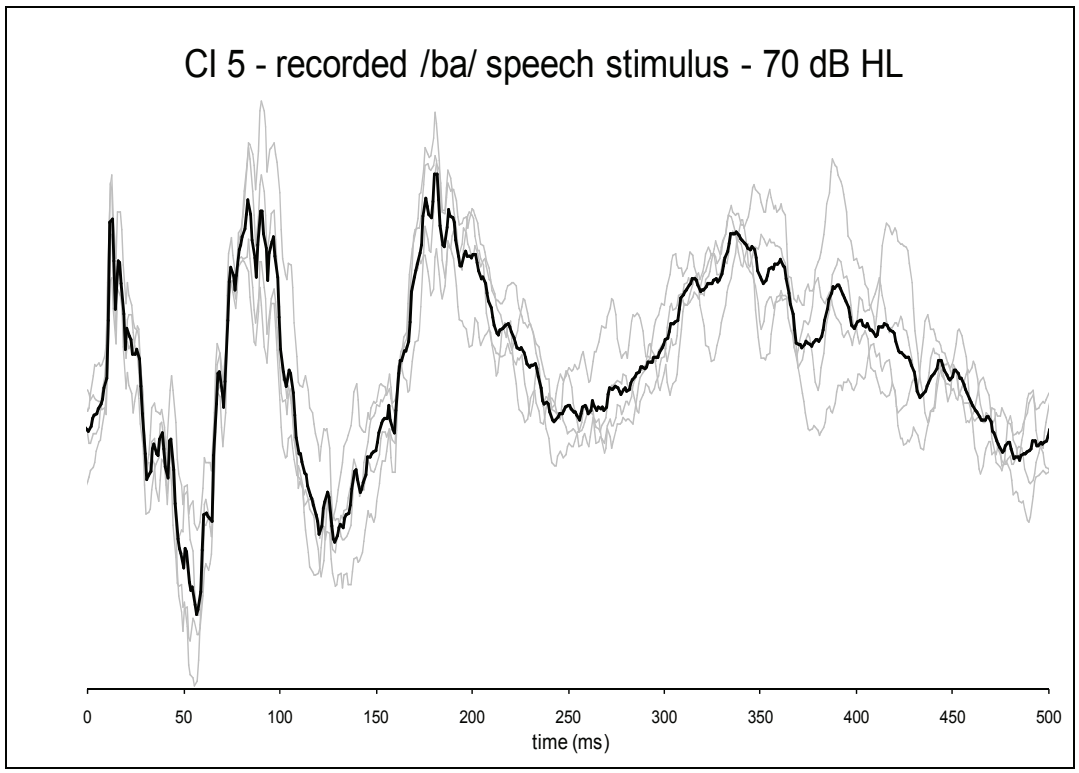

CI 3 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

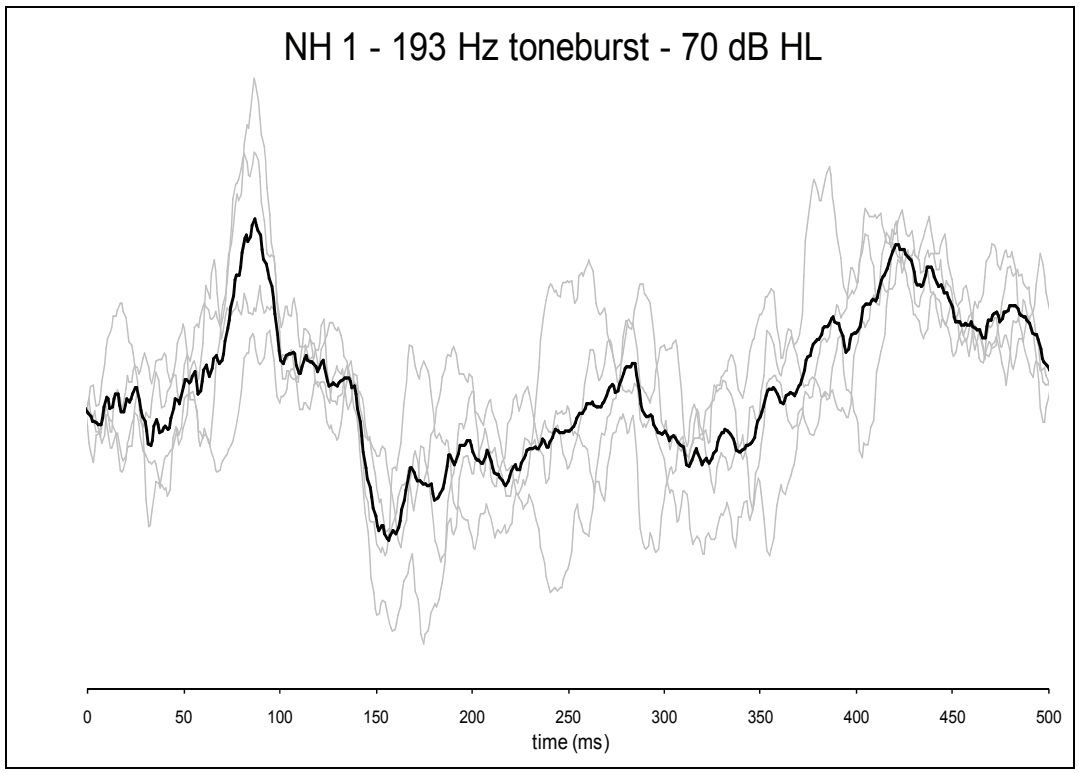

CI 3 - recorded /ba/ speech stimulus- 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

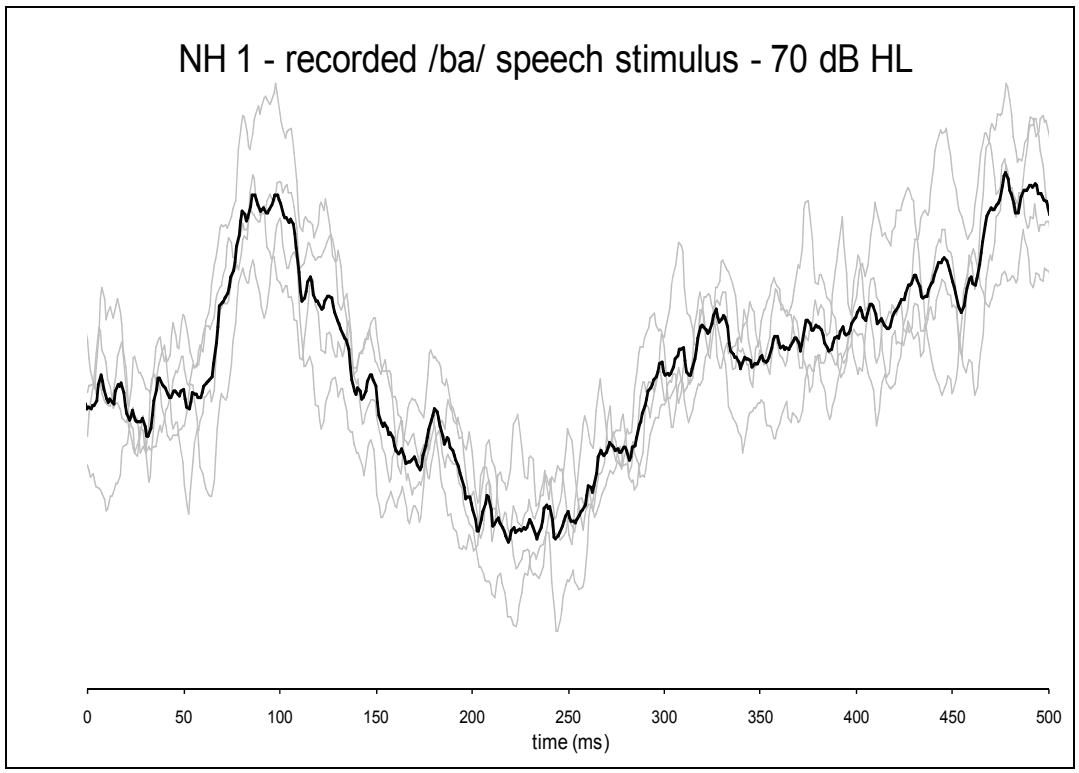

CI 4 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

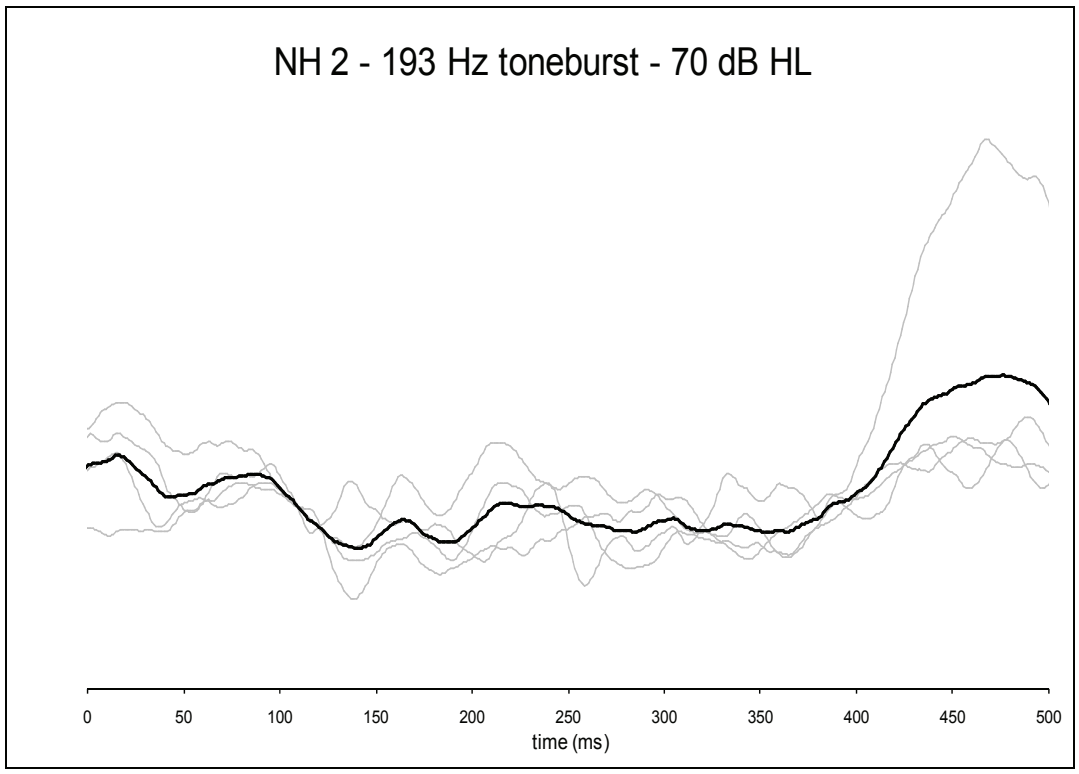

CI 4 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

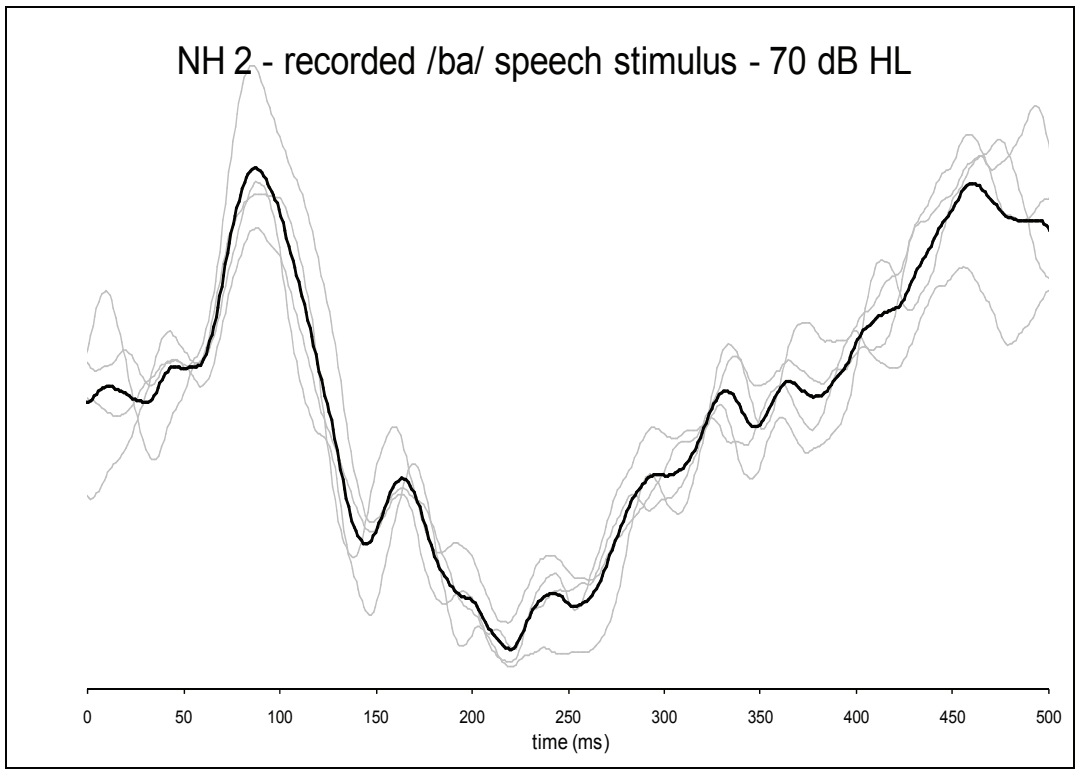

CI 5 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

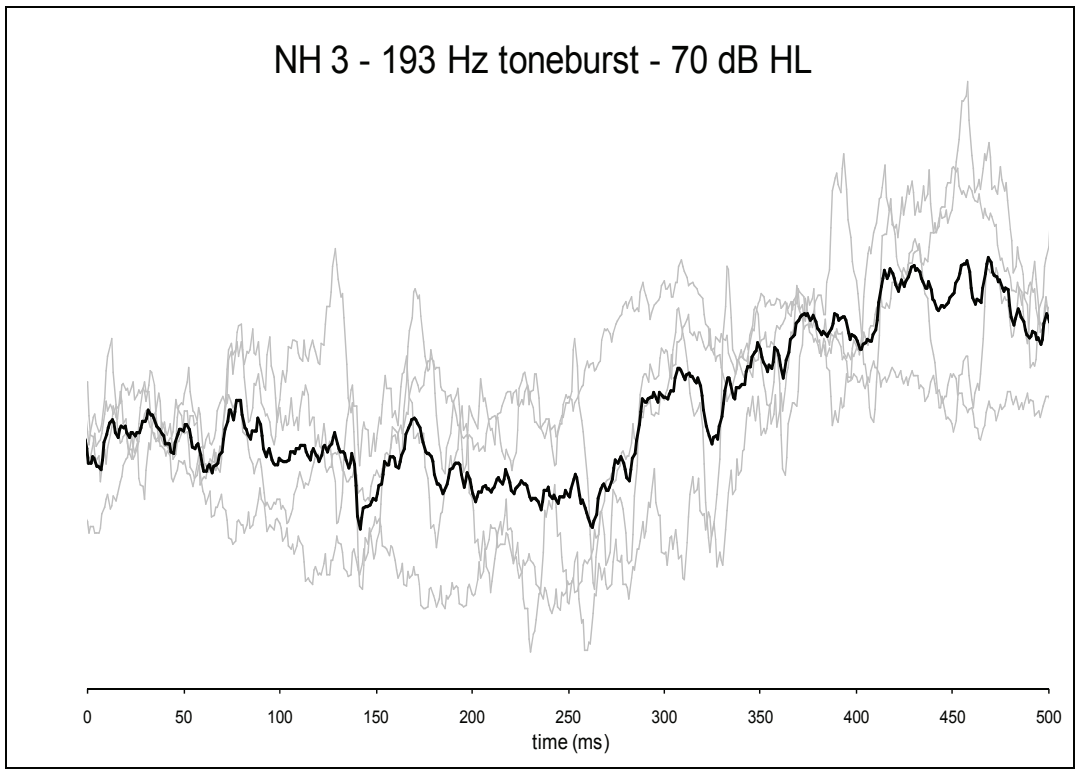

CI 5 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

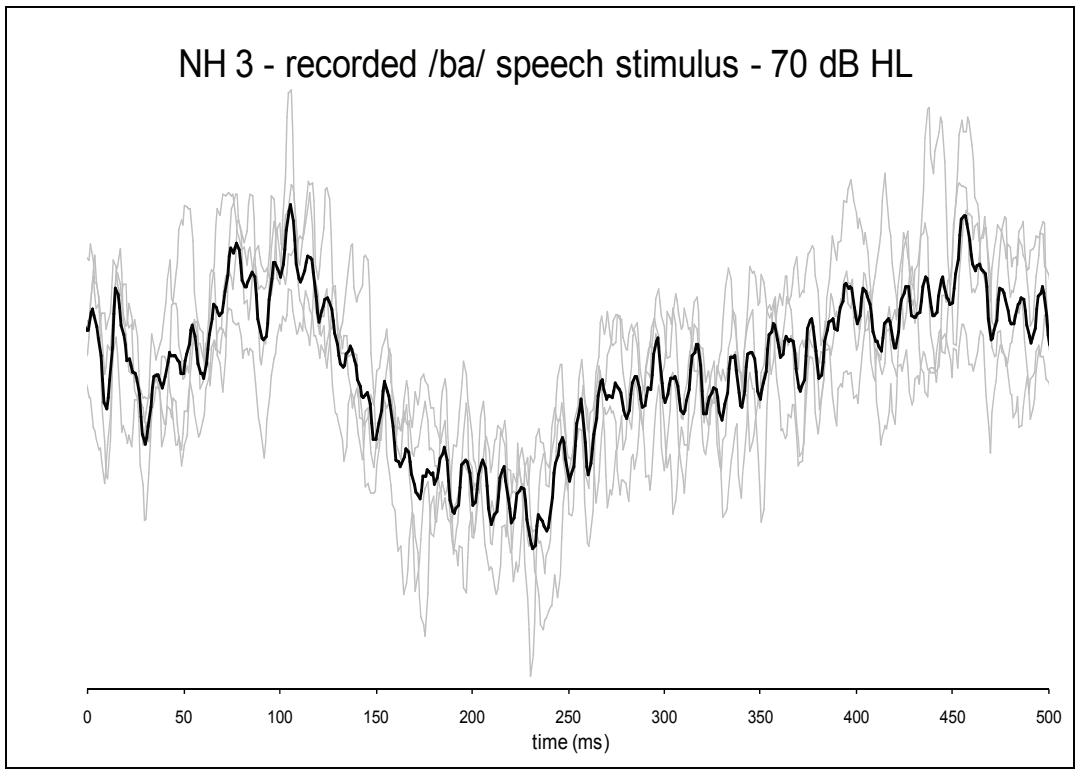

NH 1 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

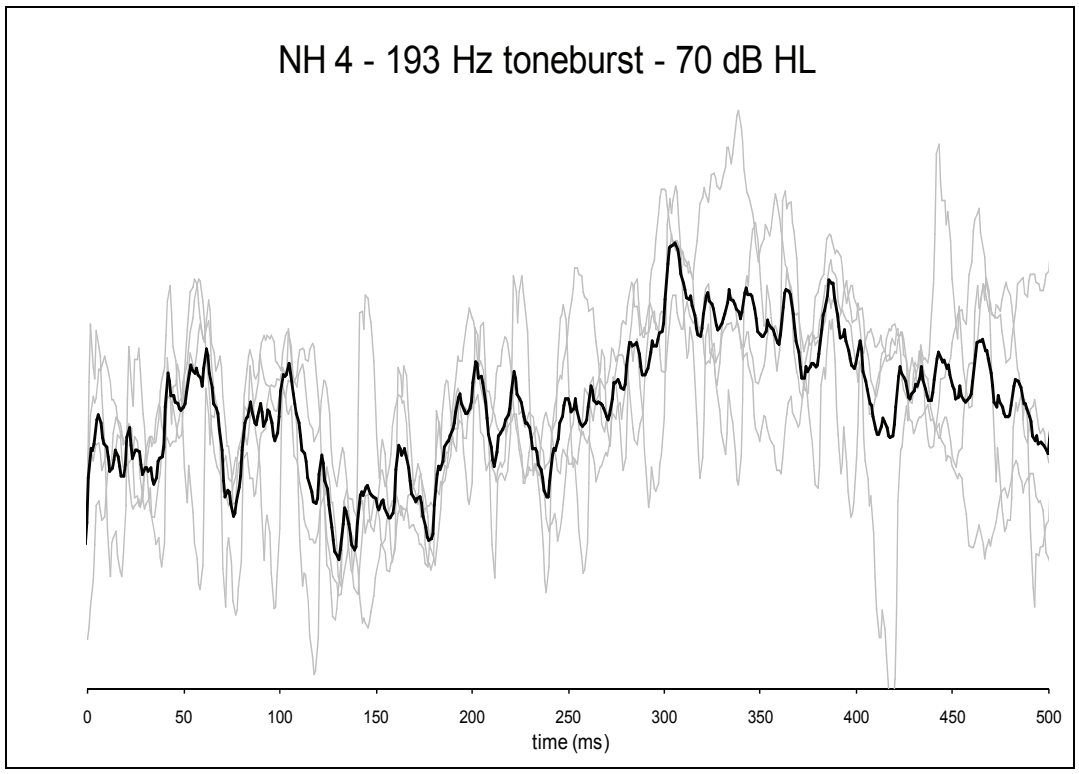

NH 1 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

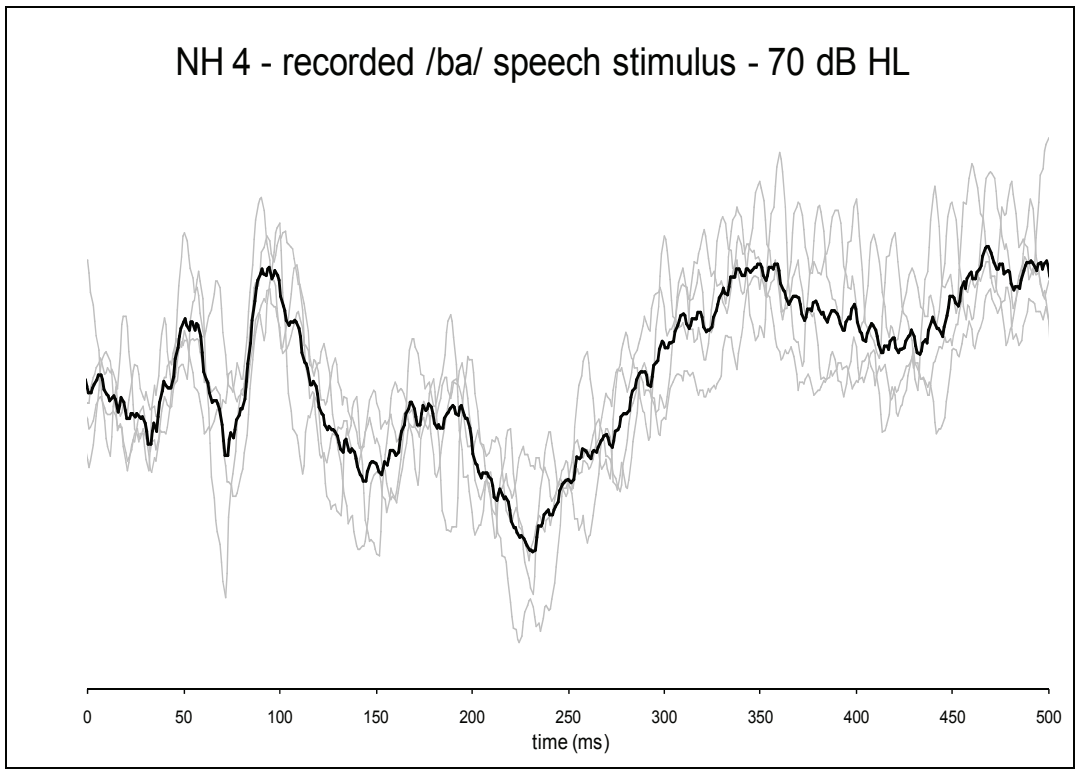

NH 2 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

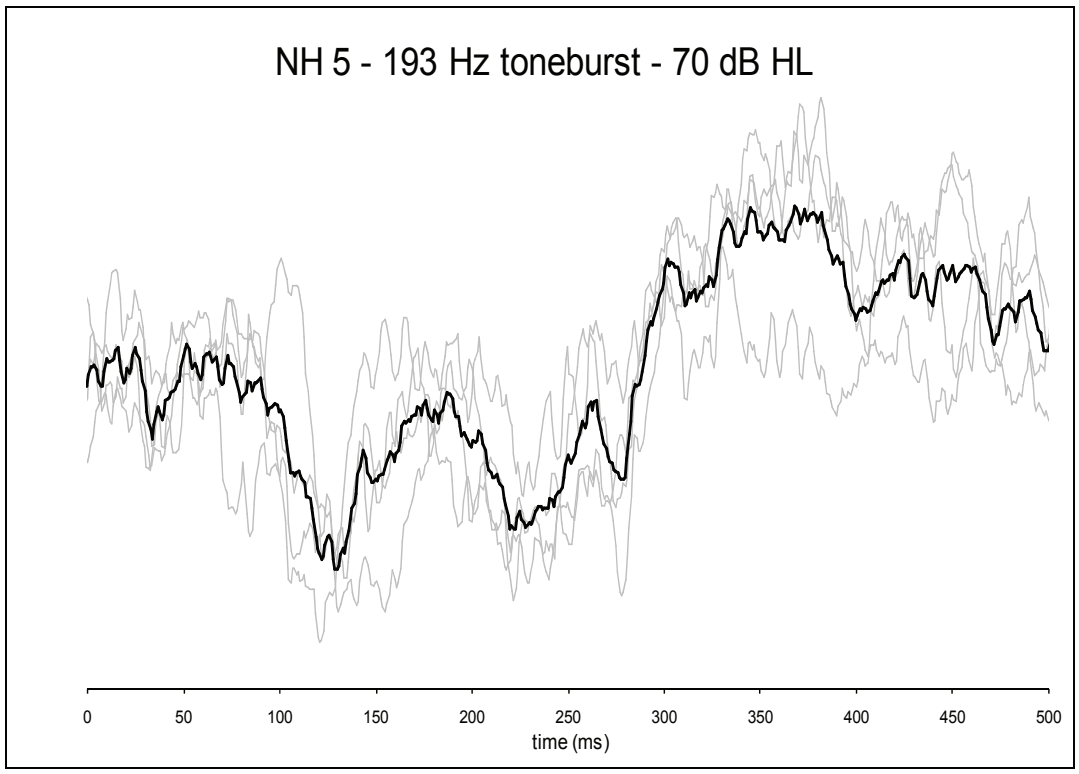

NH 2 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

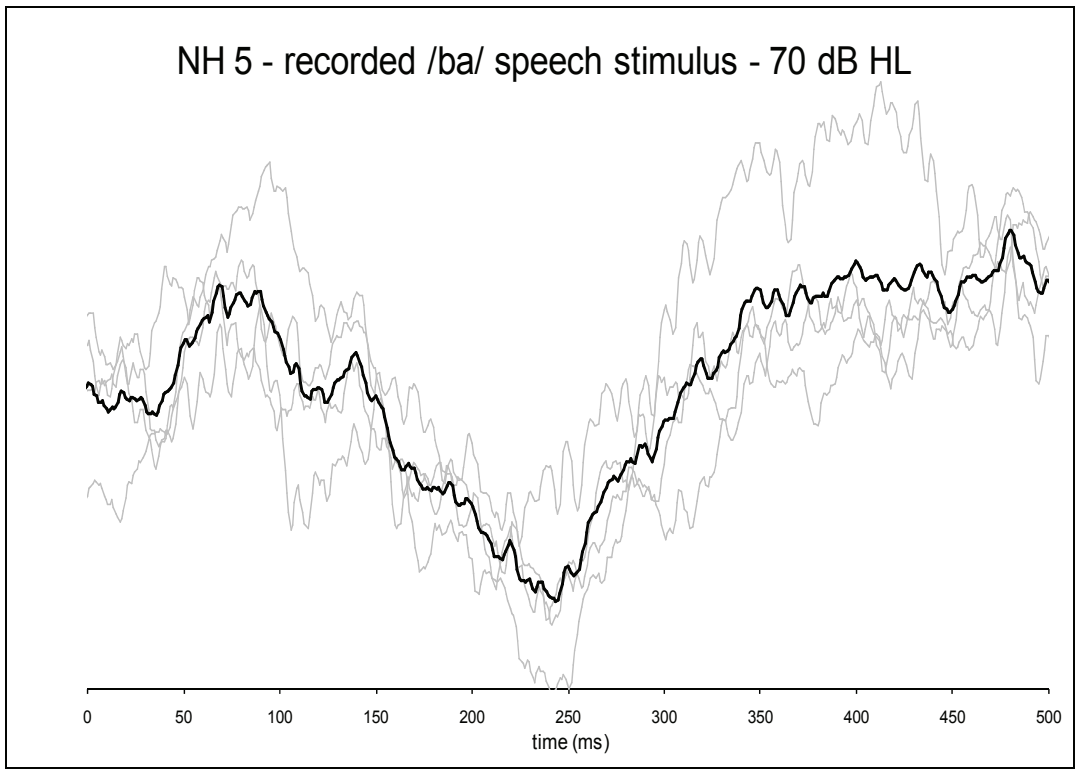

NH 3 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

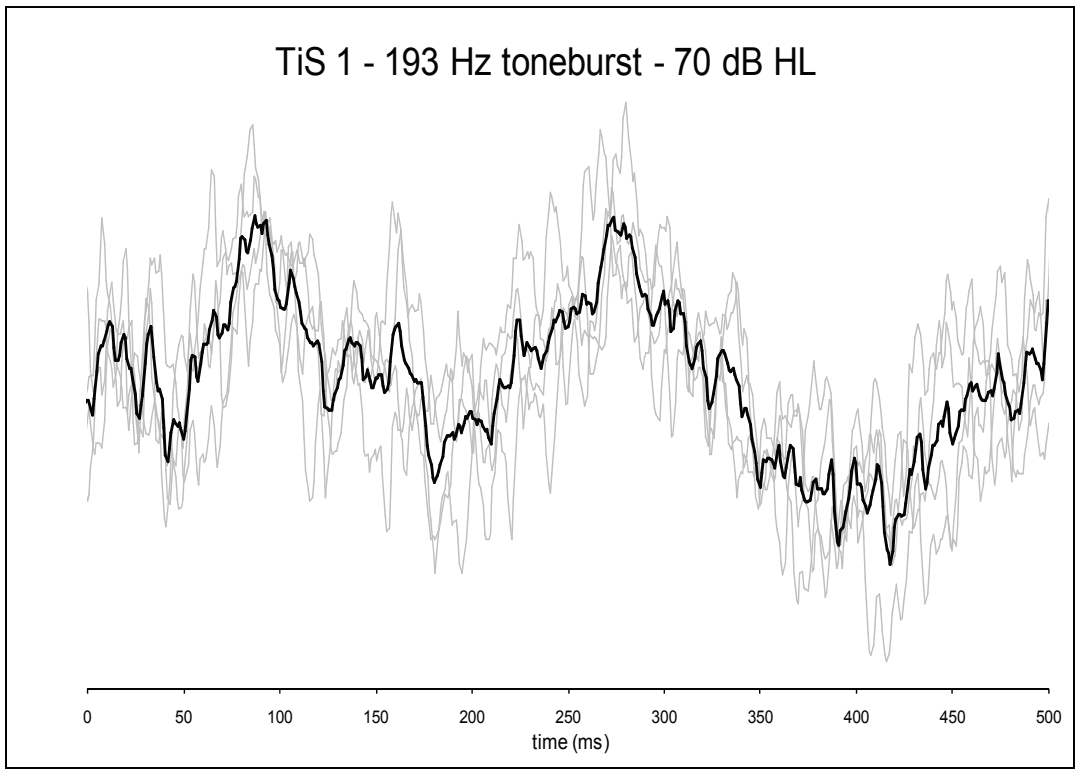

NH 3 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

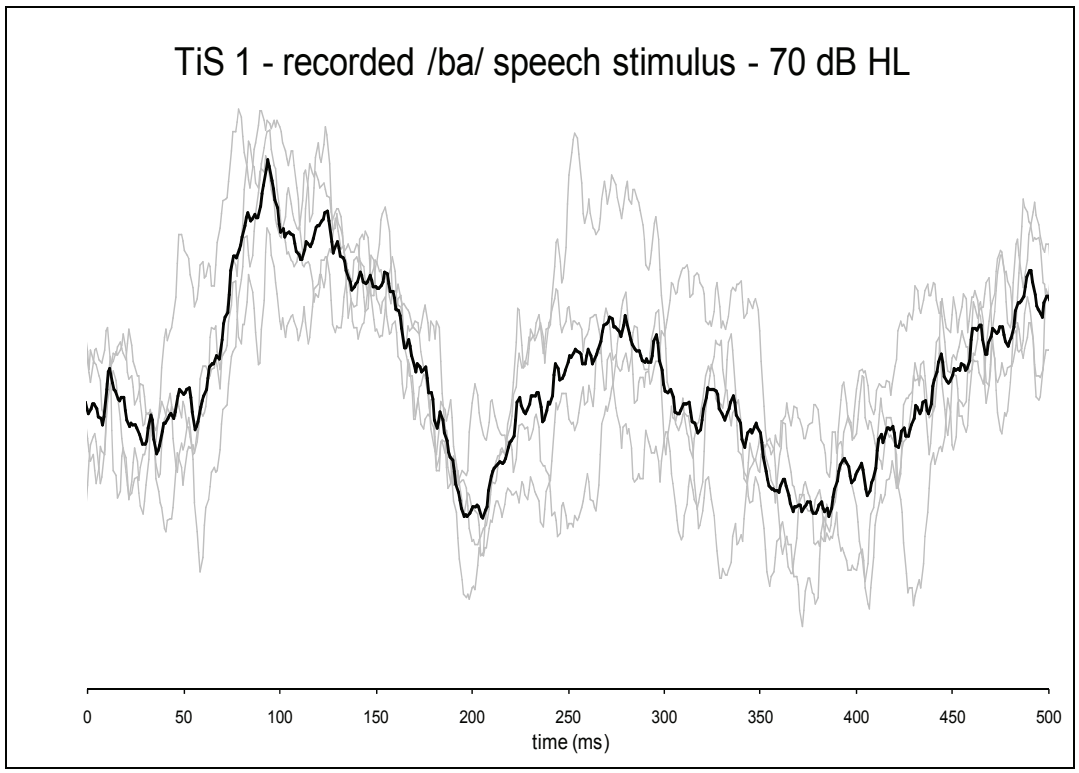

NH 4 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

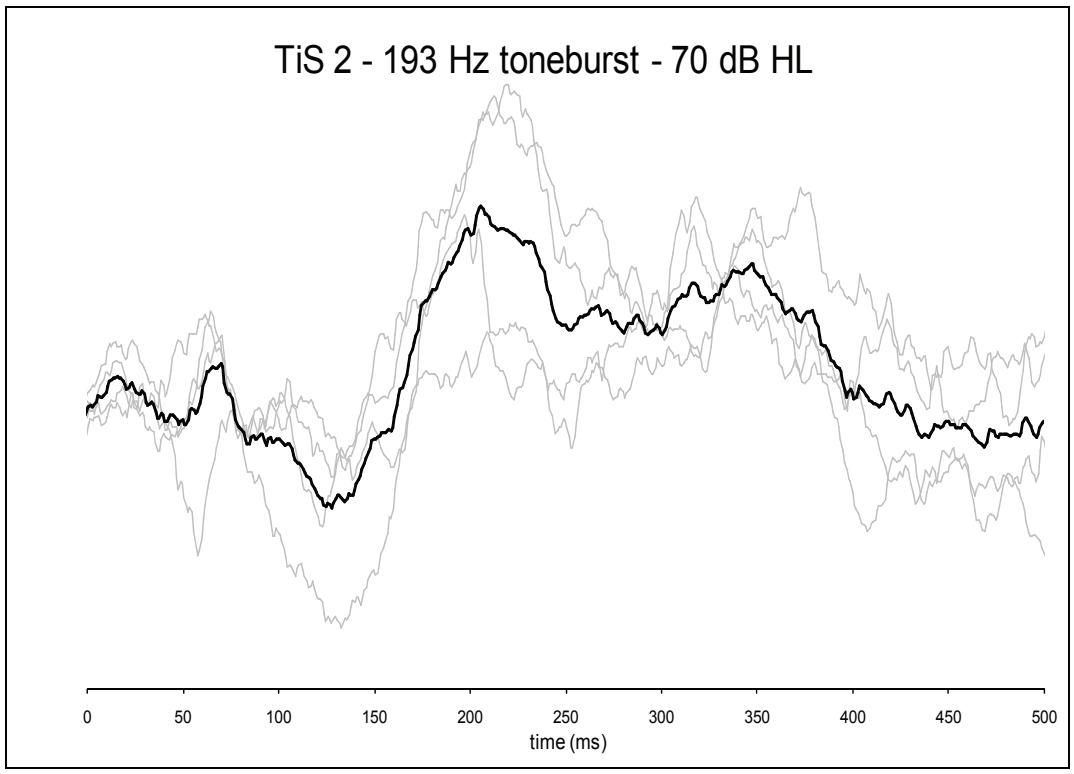

NH 4 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

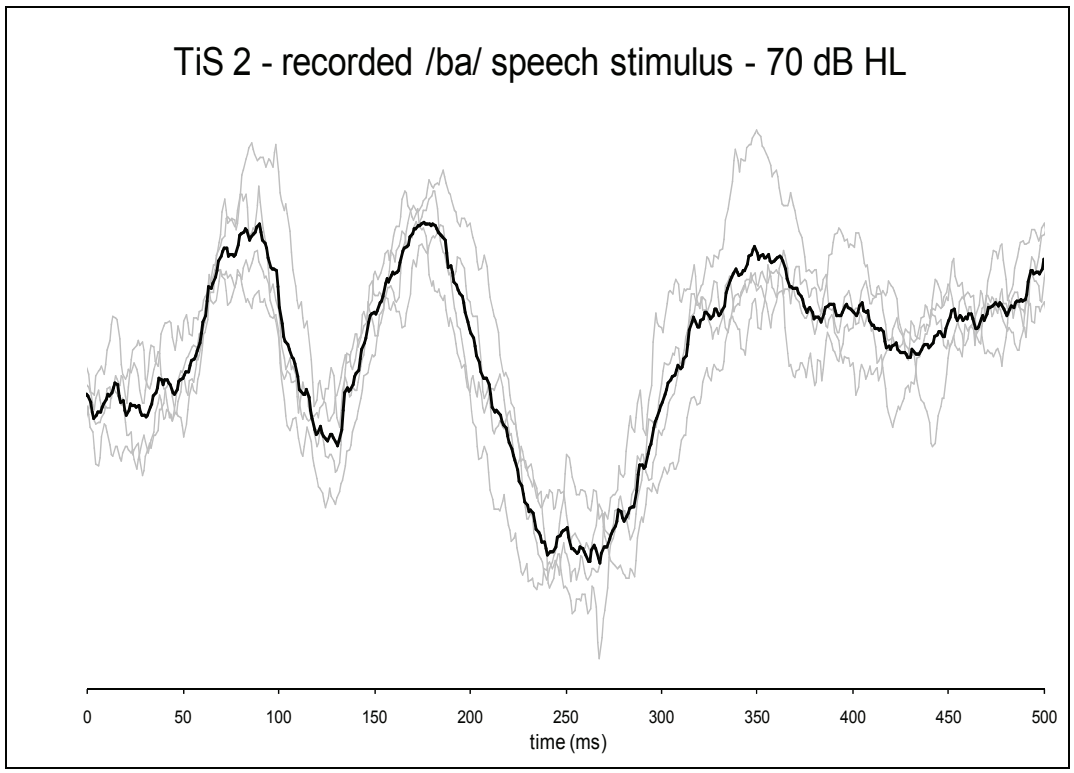

NH 5 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

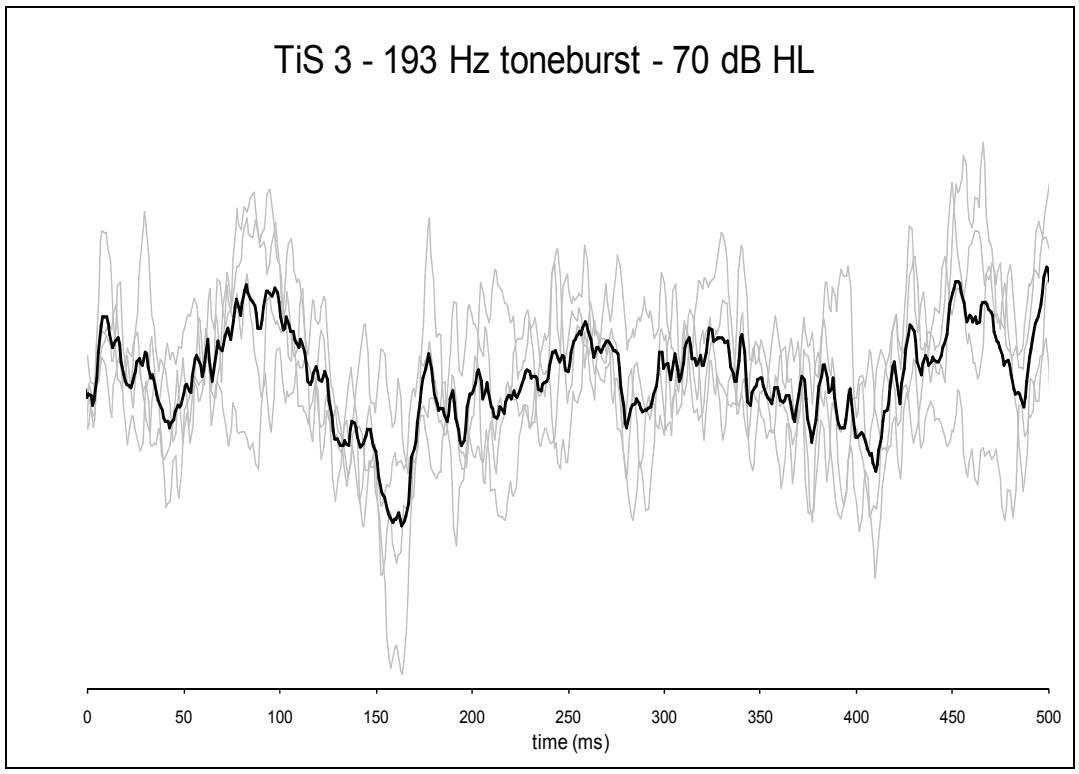

NH 5 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

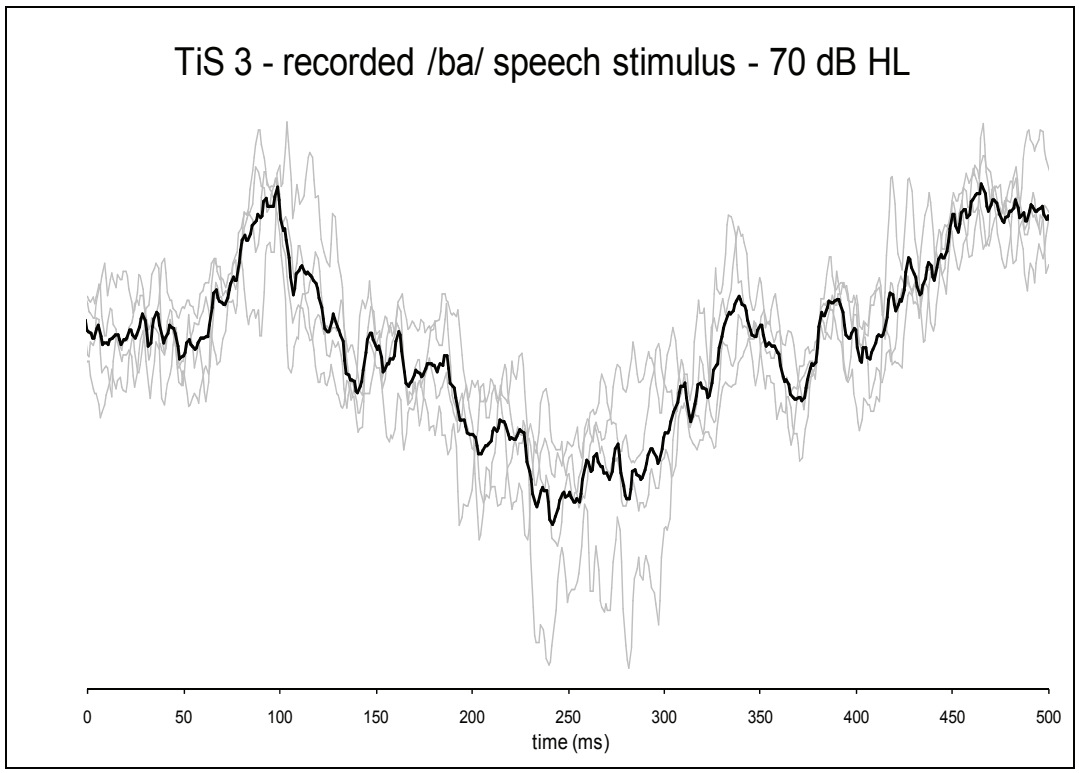

TiS 1 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

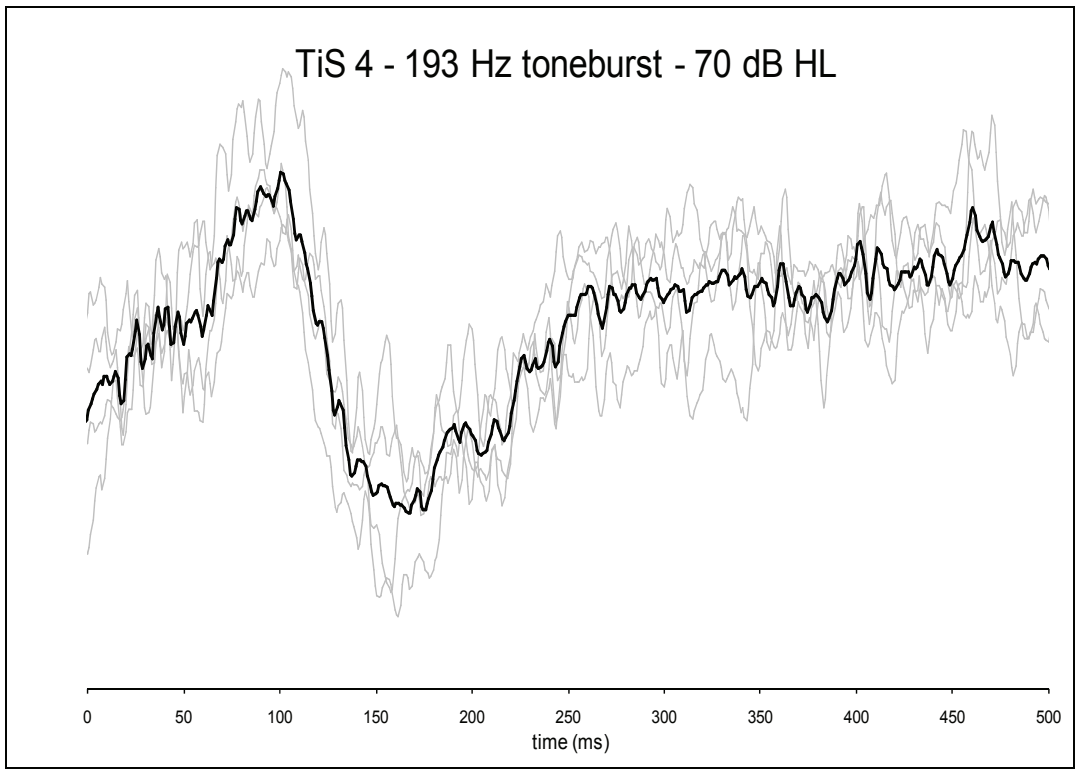

TiS 1 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

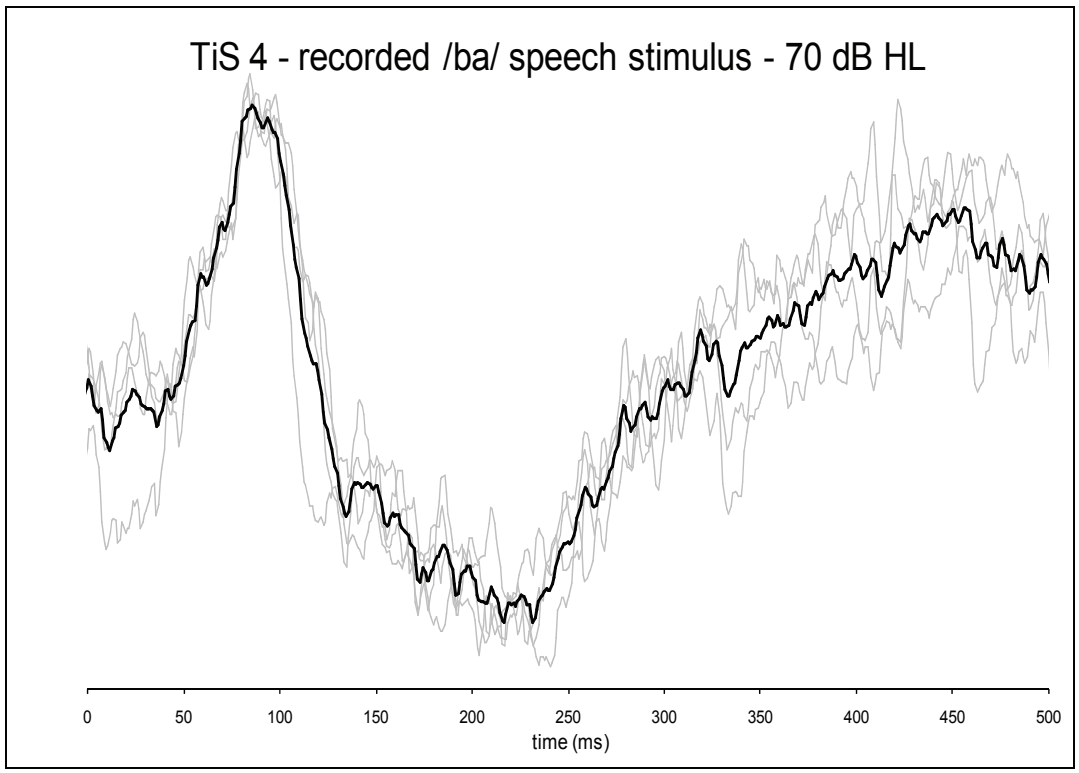

TiS 2 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

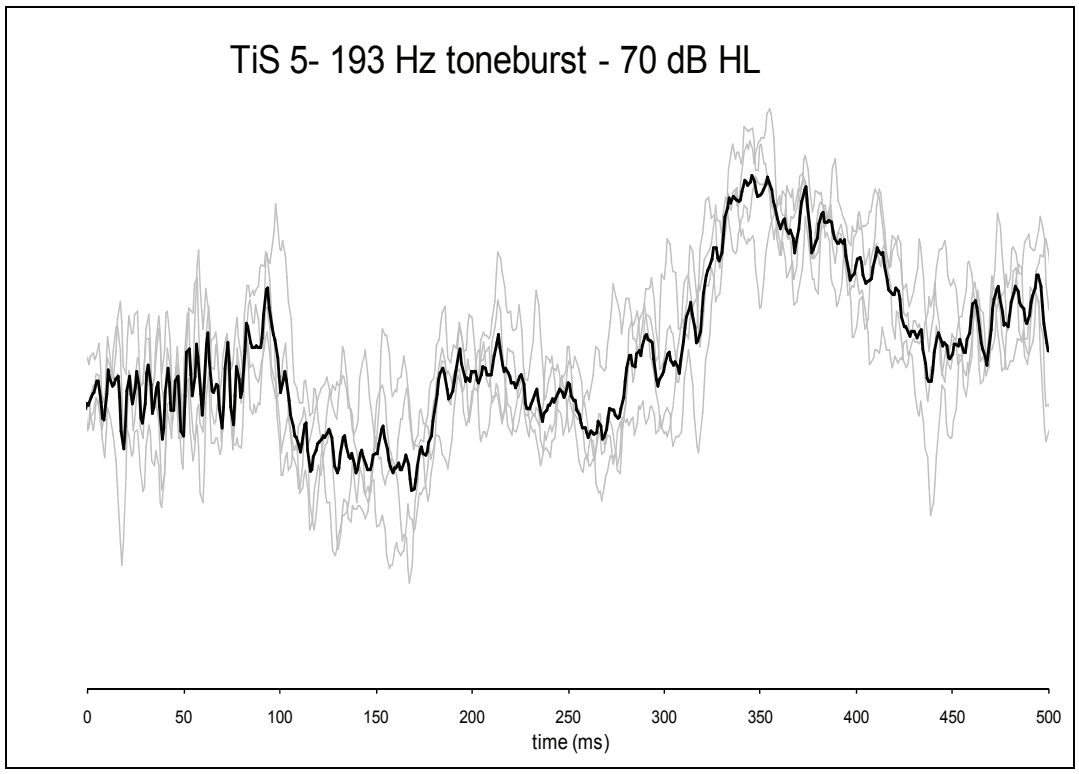

TiS 2 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

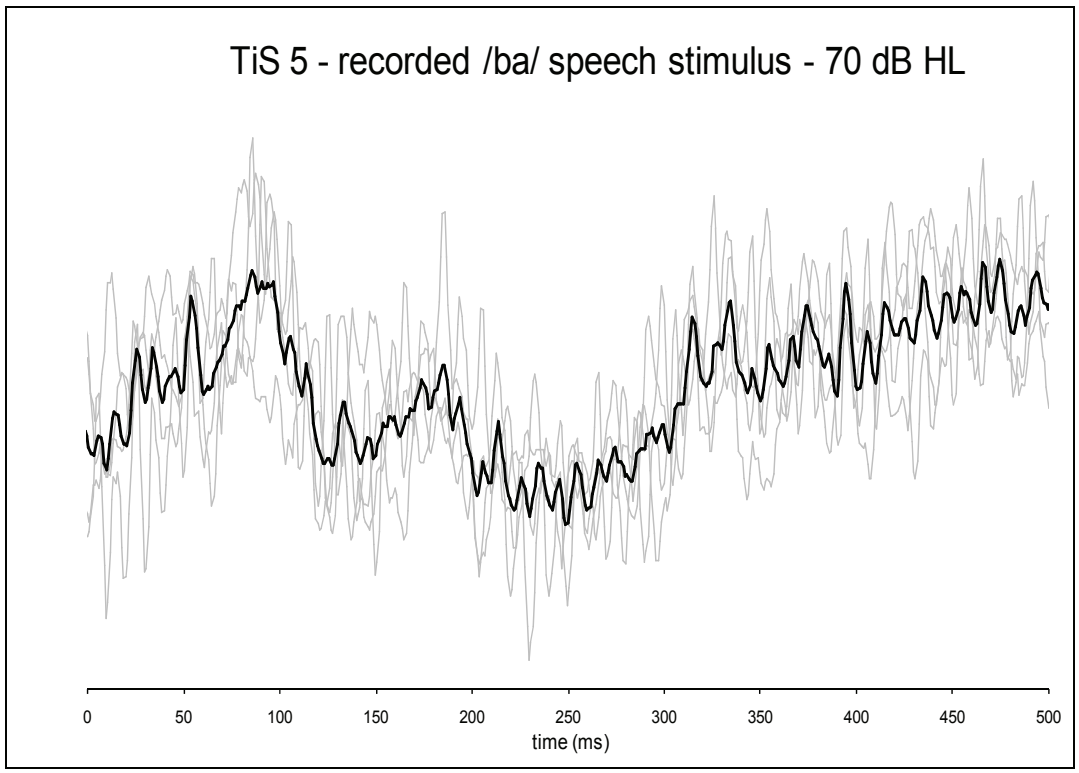

TiS 3 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

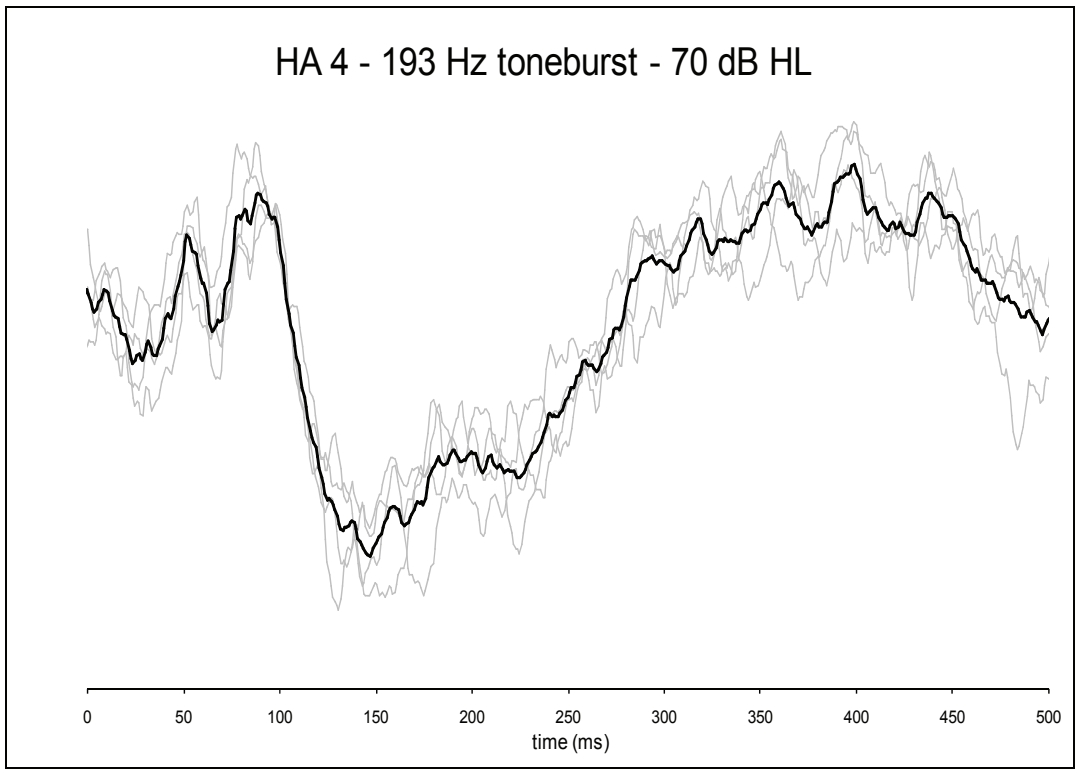

TiS 3 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)

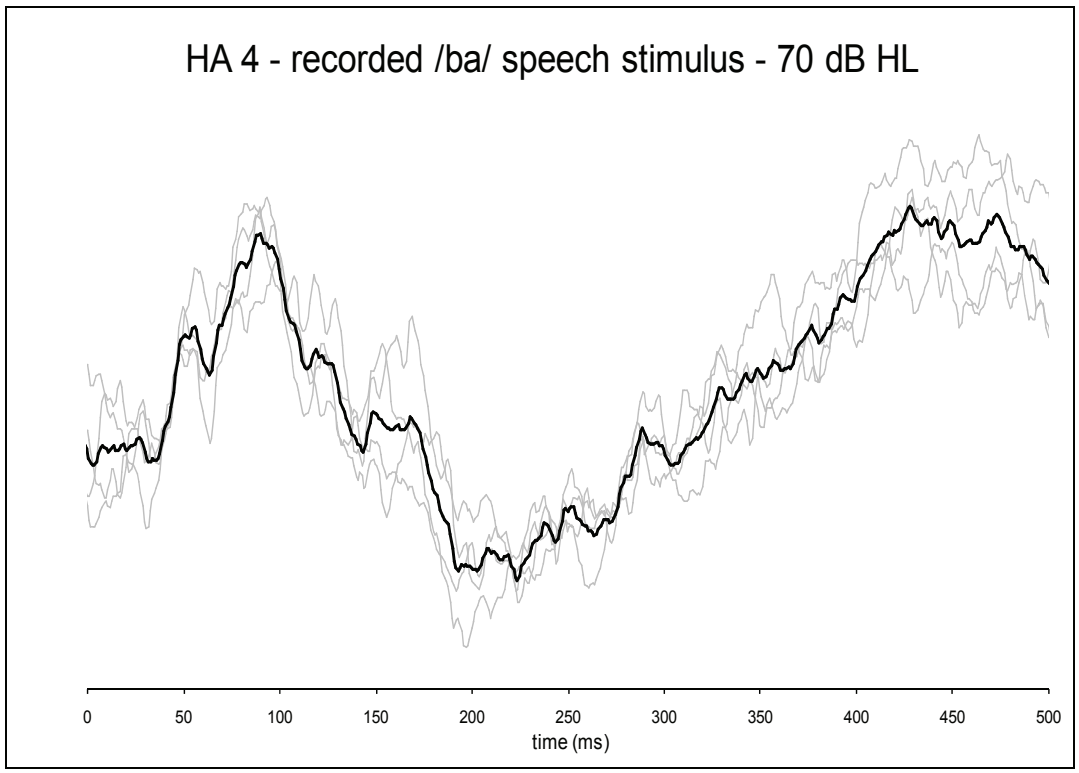

TiS 4 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)


TiS 4 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)


TiS 5- 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)


TiS 5 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)


HA 4 - 193 Hz toneburst - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)


HA 4 - recorded /ba/ speech stimulus - 70 dB HL
0
50
100
150
200
250
300
350
400
450
500
time (ms)